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THE PRINCIPLES AND PRACTICE OF
DERMATOLOGY

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THE
PRINCIPLES AND PRACTICE
OF
DERMATOLOGY

DESIGNED FOR STUDENTS AND PRACTITIONERS

BY
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*WITH FIVE PLATES, ONE IN COLOR, AND THREE HUNDRED AND
EIGHTY-FOUR TEXT ILLUSTRATIONS*

SECOND EDITION

NEW YORK AND LONDON
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1911

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TO MY FATHER

DR. ROBERT BURNS PUSEY

WHOSE MEMORY IS AN

INSPIRATION

95189

PREFACE TO SECOND EDITION

THE preparation of this edition has given me the opportunity to embody in the work the recent additions to dermatological knowledge. Among new topics which have been considered are: Gangosa, Sporotrichosis, Tinea intersecta, Tinea albiginea, Brown-Tail Moth Dermatitis, Dermatitis from Straw-Mites, Cestode Larva in Skin, Cutis plicata, Cutis verticis gyrata, Dermolysis, Paraffinoma, Multiple Hereditary Telangiectasia, and Trichonodosis. I have also written more fully on the therapeutic uses of radium, and of refrigeration with liquid air and solid carbon dioxide, on pellagra and yaws, and particularly on the epochal additions to our knowledge of syphilis which have come from the study of experimental syphilis in monkeys and apes, the discovery of the spirocheta pallida, and the application of the serum complement reaction test to the diagnosis of syphilis. The mere enumeration of these very recent additions to our knowledge indicates the activity of present-day dermatology. I have collected in a bibliography at the end of the book references to timely or very important articles, and otherwise considerably increased the Bibliography, and have very much enlarged the Index. Through the generosity of my publishers, I have been able to add numerous new illustrations.

These additions constitute the changes in the work, for fortunately I have not found it necessary to make many emendations or changes. A few corrections have been made.

I trust it is unnecessary for me to say that I am grateful for the generous reception which the book has had.

WILLIAM ALLEN PUSEY.

72 MADISON STREET, CHICAGO.

PREFACE

THE following pages represent an attempt to present the subject of dermatology, comprehensively but as concisely as practicable, and if the undertaking is successful the work is a reflex of current dermatological knowledge. But no one can review the subject of dermatology without meeting topics concerning which he must write upon the basis of his individual judgment—sometimes in the face of other and weightier opinions; in such instances I have not hesitated to accept the responsibility of the position, not, however, I trust, without making due statement of other views.

I have given somewhat more space than usual to the principles of dermatology—the anatomy and physiology of the skin, general etiology, pathology, symptomatology, and treatment of diseases of the skin. This has been done because it is highly desirable in any study to be as fully acquainted as possible with the fundamental knowledge of the subject; it is desirable not only in order to obtain a satisfactory grasp of the subject, but in order to make its study interesting. Because of its practical importance, general treatment especially has been considered in full.

I am indebted for aid of one sort and another to many friends, particularly among my colleagues in Chicago and in the American Dermatological Association. I am under especial obligations to Dr. C. J. White, of Boston, to Dr. Grover W. Wende, of Buffalo, to Dr. J. F. Schamberg and Dr. M. B. Hartzell, of Philadelphia, to Dr. M. F. Engmann, Dr. W. H. Mook, and Dr. Joseph Grindon, of St. Louis, to Dr. J. A. Fordyce and Dr. S. Pollitzer, of New York, to Dr. J. M. Winfield, of Brooklyn, and to Dr. M. L. Heidingsfeld, of Cincinnati, for placing at my disposal large collections of illustrations from which I have drawn freely. For aid in reviewing literature and in correcting manuscript I am indebted to my associates, Dr. R. W. McClintock and Dr. Frederick G. Harris. Finally, for much patient help in various ways, I am indebted to my wife.

William Allen Pusey.

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ABBREVIATIONS

Duhring = "Cutaneous Medicine," L. A. Duhring; J. B. Lippincott Co., 1895.

Morrow = "A System of Genito-Urinary Diseases, Syphilology and Dermatology," edited by Prince A. Morrow; D. Appleton & Co., 1894.

Hebra = "Diseases of the Skin," by Ferdinand Hebra, Fagge's translation; The New Sydenham Society, London, 1865.

Kaposi = "Diseases of the Skin," by M. Kaposi, Johnston's translation; Wm. Wood & Co., 1895.

Crocker = "Diseases of the Skin," by H. Radcliffe Crocker; P. Blakiston's Son & Co., 1903.

Stelwagon = "Diseases of the Skin," by Henry W. Stelwagon; W. B. Saunders & Co., 1905.

Hyde and Montgomery = "Diseases of the Skin," by James Nevins Hyde and Frank Hugh Montgomery; Lee Bros. & Co., 1904.

Bangs and Hardaway = "Text-book of Genito-Urinary Diseases, Syphilis, and Diseases of the Skin," edited by L. Bolton Bangs and W. A. Hardaway; W. B. Saunders, 1898.

MacLeod = "Pathology of the Skin," by J. M. H. MacLeod; P. Blakiston's Son & Co., 1903.

La Pratique = "La Pratique Dermatologique," Besnier, Brocq & Jacquet, Editors, Masson et Cie, Paris, 1900.

Unna's Histopathology = "Histopathology of Diseases of the Skin," by P. G. Unna, Walker's translation; Macmillan & Co., 1896.

International Atlas = "International Atlas of Rare Skin Diseases"; Leopold Voss, Hamburg und Leipzig.

Jour. Cutan. Dis. = *Journal of Cutaneous Diseases*, New York.

Brit. Jour. Derm. = *British Journal of Dermatology*, London.

Archives = *Archiv. für Dermatologie und Syphilis*, Vienna.

Annales = *Annales de Dermatologie et de Syphiligraphie*, Paris.

Monatshefte = *Monatshefte für praktische Dermatologie*, Hamburg.

THE PRINCIPLES OF DERMATOLOGY

SECTION I

THE PRINCIPLES OF DERMATOLOGY

I.–ANATOMY [1]

Macroscopic Characteristics of the Skin.—The skin, or general integument (*integumentum commune*), covers the external surface of the body and is continuous at the natural orifices with the mucous membranes. At the lips, eyelids, and urinary meatus, the transition from skin to mucous membrane is abrupt; at the other orifices it is gradual. The entire area of the skin of an individual of average size is about one and one half square meters. The general characteristics of the skin, while uniform in their larger features, vary with races, individuals, age, sex, and, to some extent, with climates and seasons and the habits of the individual as regards exposure to the elements and mode of life.

Tension.—As would be expected from its close adaptation to the underlying parts, the skin is normally in a condition of moderate tension. This is evident in the shrinking of a piece of skin immediately after excision, and in the retraction of the skin when an incision is made through it. Over most of the surface of the body the skin rests on a cushion of subcutaneous fat, to which it is intimately attached, but which permits of free movement. The skin of the scalp and glans penis is bound down to the underlying tissues more firmly than in other locations. Abnormal outward pressure in pregnancy, tumors, œdema, and other swellings increases the tension of the skin, even to the point of overdistention, with laceration of the fibers of the deeper layers and the formation of atrophic scars when the pressure is relieved.

Elasticity.—The elasticity of the skin, while only slight, is almost perfect: that is, after stretching, it at once completely, or almost completely, regains its original shape. This is not due to the elastic fibers, as will be explained later.

Thickness.—The thickness of the skin varies greatly in different parts of the body, and somewhat in different individuals. Exclusive of the subcutaneous tissue, which varies in thickness enormously according a the deposit of fat in it is small or great, the skin is from .5 mm. t 8 mm. thick, being generally thin on flexor and thick on extensor su faces. It is thinnest on the eyelids and the prepuce—parts which hav the freest motion—and thickest on the palms, soles, and buttocks—part which are subjected to greatest pressure and friction—and at point which serve as insertions for muscles, as the lips and sides of the nos

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[1] MacLeod, "Pathology of the Skin."—Unna, v. Ziemssen's "Handbook."—Duhring, "Cutaneous Medicine."—Elliot, Article on "Anatomy of the Skin." "Reference Handbook of the Medical Sciences," Wm. Wood & Co.—Szymonowicz and MacCallum, "Histology."

Strength and Texture.—The tensile strength of the skin is conside able, but varies with its thickness. According to Sappey, a strip o skin 3 mm. long and 10 mm. broad sustained a weight of 12 kil (26 lbs.).

The unctuous, velvety feel of the skin is due, first, to its fine te ture, and, after that, to its slight greasiness and the abundant growt of delicate hair which covers it. The lubrication of the skin, which universal over its surface, is due partly to the secretion of fat by t sweat and sebaceous glands and partly to the formation of fat in t cells of the horny epidermis. The presence of this fat increases the i permeability of the skin.

Color.—The color of the skin varies with age, sex, and individu peculiarities, with race, and to some extent with climatic condition The color in the dark races is due to the large amount of pigment pre ent in the epidermis. This pigment is quite opaque and absorbs t light without reflecting it. In the white races the pigment plays a muc less important rôle. The color is due rather to the slight transluce of the epidermis, which allows the red color of the corium to filt through, and to the reflection and refraction of light by the cells of t epidermis. This translucence is chiefly produced by the granular laye The variations in color of the skin in different parts of the body are d largely to variations in the amount of pigment, and to a less degree variations in vascularity. The deposit of pigment is greatest and t color is consequently darkest in the skin of the nipples and their areol of the axillæ, of the genitals and the perineum, and around the anu The differences in color due to vascular variations are chiefly appare in the flush area of the face—the parts affected in blushing—where t blood supply of the skin is greatest and where the nervous control is mo sensitive.

Markings.—The cutaneous surface is diversified by innumerable fi lines and furrows, and by numerous coarse furrows. These coarse fu rows represent the creasing of the skin, which necessarily occurs in i various movements because of its attachment to the underlying stru tures. The character of these furrows is determined, first, by the pr dominating motion of the part, and, second, by the arrangement of t fibrous bundles of the skin and by their underlying attachments. Th direction of the furrows is in a general way transverse to the plane of motion of the part.

The coarse furrows occur where the range of motion is greatest, and their depth and direction are determined chiefly by the attachment of the fibrous tissue of the skin to the underlying structures. Coarse furrows of moderate degree are found over the knuckles, the flexor surfaces of the joints of the fingers and toes, and on the palms and soles. Still deeper furrows are those over the extensor surfaces of the knees and elbows. The most marked are the inguinal folds, due to the attachment of the skin to Poupart's ligaments, the gluteal fold, the fold between the buttocks and thighs, and the popliteal folds. Of the same character as these folds are the wrinkles which occur on the face in the expression of the emotions. These are due to the attachment of the skin to the muscles of expression, which cause wrinkling when they contract. Of the same character, also, are dimples. In old age, and often in disease, the furrows of the skin become exaggerated from shrinkage in the amount of subcutaneous fats.

The fine furrows mark the entire surface of the body into innumerable small, rhomboid, or lozenge-shaped figures. A good example of the pavement-like marking of the skin that is produced by the fine furrows is seen on the flexor surfaces of the wrists.

PAPILLARY RIDGES AND FURROWS.—The finest markings of the skin are the minute papillary ridges with the depressed line between, which are seen upon the palms and soles. These papillary ridges owe their existence to the fact that the papillæ on these surfaces are large and are arranged in parallel rows, thus forming ridges. Two rows of papillæ are usually found on one ridge, and in a line between these rows on the crest of the ridge are the minute openings of the sweat ducts. These markings are most apparent upon the flexor surfaces of the distal phalanges of the fingers and toes, where they form peculiar whorls, but they are visible over the entire plantar and palmar surface. As is well known, these markings are fixed and for the individual distinctive. (Fig. 1.)

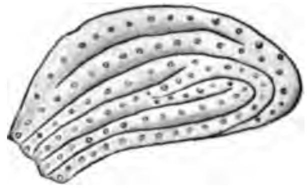

FIG. 1.—PAPILLARY RIDGES FROM THE FINGER-TIP, SHOWING OPENINGS OF SWEAT DUCTS.

Pores.—The pores are the openings of the follicles upon the surface of the skin. The sebaceous glands and the hair follicles usually have common openings, and the depressions representing their openings are visible over the entire body. The largest pores are those of the sebaceous glands of the face. They vary considerably in size in different individuals. In skins of fine texture their openings may be visible only upon close examination, but when their size is large they form distinct conical depressions, especially upon the alæ nasi. Sweat pores are ordinarily invisible to the naked eye, but with a magnification of two or three diameters they can be seen upon the palms and the soles (Fig. 1).

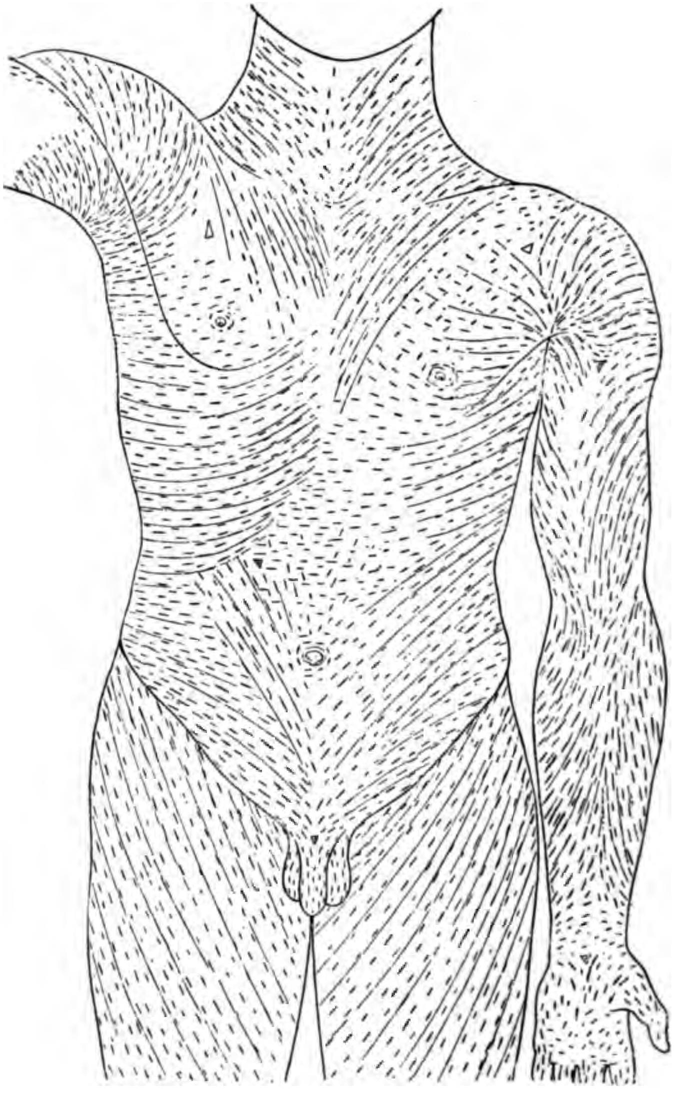

FIG. 2.—CLEAVAGE OF THE SKIN. (C. Heitzmann, Duhring's Cutaneous Medicine.)

Cleavage.—The term cleavage of the skin has reference to the direction in which the skin tears. This is determined by the arrangement of the fibrous bundles, which constitute the chief strength of the skin.

Dupuytren, from his observations on punctured wounds in war, first called attention to the fact that the skin at any given point has a tendency to split in a definite direction. Later, Langer mapped out the lines of cleavage over the entire body by observing the direction in which tearing occurred when the skin was punctured with an awl. He established the fact that the lines of cleavage were definite and correspond in direction, as do the furrows of the skin, with its lines of greatest tension. (Fig. 2.)

Microscopic Anatomy of the Skin.—The skin consists of two essentially distinct layers:

(1) The epidermis.

(2) The corium and the subcutaneous tissue.

Embryology.—These two layers are of different embryonic origin, and their embryonic difference is sharply emphasized by the differences in their structure and by the sharp line of demarcation between them. The epidermis is formed from the ectoderm, the derma from the mesoderm; and the two layers in their histological characteristics and in their pathological variations show the contrasting features of epiblastic and mesoblastic tissues.

At the end of the first and the beginning of the second month of fœtal life the epidermis presents one or two layers of cuboidal cells, and, beneath these, a row of very fine cells which represent the first trace of the mucous layer. By the fourth month the upper cells have become somewhat flattened and more granular, and begin to approximate in character the horny layer, while the lower very fine cells, having proliferated rapidly, present a layer of more nearly typical epithelial cells. By the sixth month the upper cells have formed a sufficiently firm horny layer to offer considerable resistance to the upward pressure from the proliferation of the cells of the mucous layer. The horny layer is not completely developed at birth, but during the later months of fœtal life the superficial cells are partly cornified and by their exfoliation and admixture with the secretions of the skin form the vernix caseosa. The first traces of the development of the hair follicles, the sebaceous glands, and the sweat glands appear from the third to the fifth months as down-growths of the cells of the mucous layer of the epidermis into the corium (the hairs at the end of the third month and the beginning of the fourth month, the sebaceous glands from the fourth

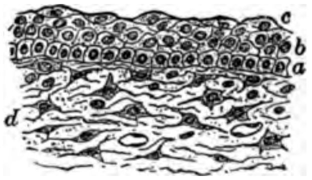

Fig. 3.—Section of Developing Skin from Human Skin of Three and a Half Months. *a*, Layer of cuboidal cells representing rete mucosum; *b*, polyhedral cells forming superficial layers; *c*, outermost flattened cells, probably the remains of the epitrichial layer; *d*, mesodermic tissue forming corium (Piersol).

to the fifth month, the sweat glands on the palms and the soles in the fifth month). The first trace of the nails appears in the third month in the marking out of the nail bed and the nail fold in the stratum corneum.

The corium and the subcutaneous connective tissue are formed from a superficial layer of the mesoblast, the skin plate of Remak, and at first consist of a thin layer of round cells. These proliferate rapidly, and by the second month begin to assume a spindle shape. A little later fibrillated tissue appears, forming interlacing bundles. In the fourth month fat appears in the meshes of the network, and blood vessels and nerves begin to push up into the structure. At the same time the prolongations of the epidermis, which ultimately form the sweat glands, the sebaceous glands, and the hair follicles, are beginning to grow down into the corium (Fig. 4). Before the sixth month the line between the epidermis and the corium is plain. In the sixth month intimations of papillæ appear upon the palms and the soles, and at birth the papillary arrangement is well developed, although the development of the papillæ is not complete until after birth. At birth the only layer which is entirely complete in development is the subcutaneous tissue. The other structures, while approximating their final character, have not entirely reached their development, and the process of their evolution continues for a time after birth.

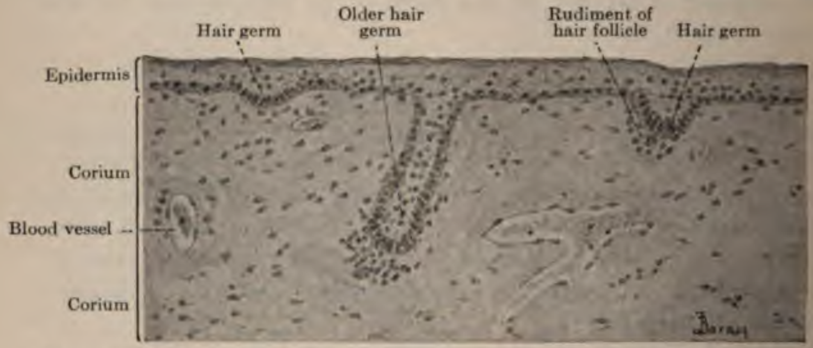

FIG. 4.—VERTICAL SECTION THROUGH THE SCALP OF A HUMAN EMBRYO OF THE FIFTH MONTH. × 230. (Szymonowicz.)

Epidermis.—The epidermis is distinctly the protective layer of the skin; it fits like a well-stretched membrane over the corium, and its most evident and, it would seem, its most important function is the production of the horny scales which compose the resistant, insensitive, and impervious coat that covers the surface of the body. It must not, however, be regarded simply as a protective membrane, for in the excretory functions of the skin the epidermis has an important part. Considering the skin as an active excretory organ, as it is, the epidermis *with its* appendages forms the parenchyma of the organ, of which the *corium* forms the stroma.

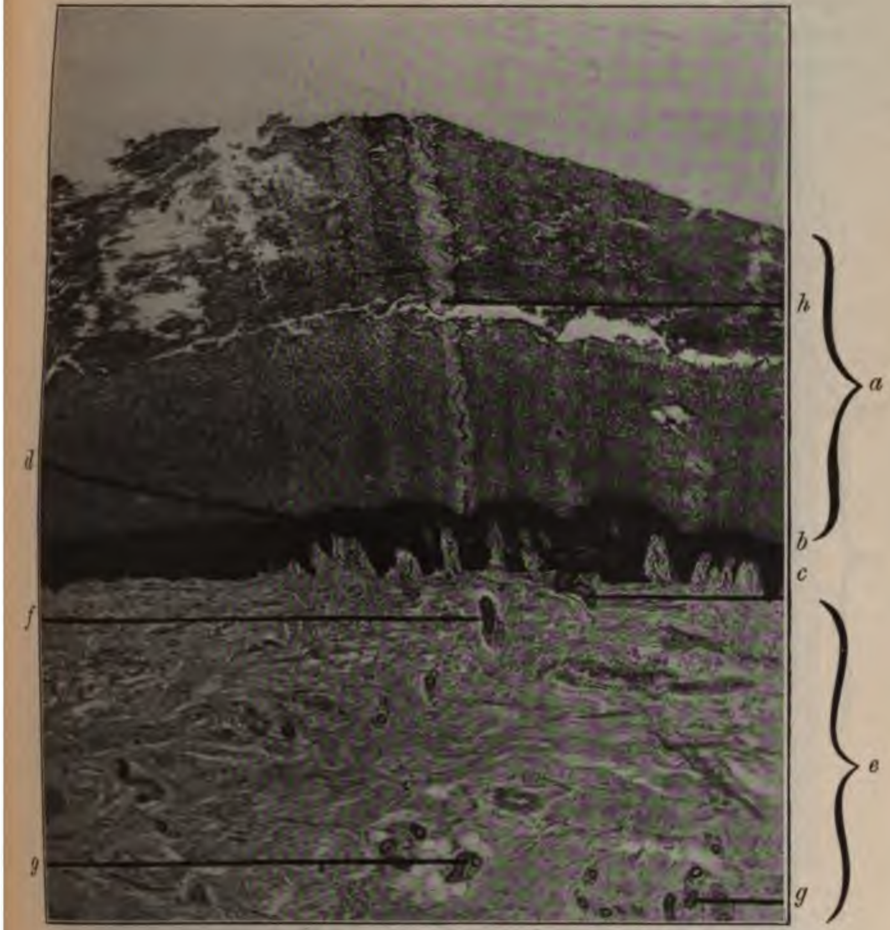

FIG. 5.—VERTICAL SECTION, SKIN OF PALM. *a*, Thick horny layer; *b*, granular layer; *c*, mucous layer; *d*, papillary layer showing normal contour in vertical section; *e*, upper part of corium; *f*, section of sweat duct in corium, the channel above through epidermis is seen; *g*, sweat ducts; *h*, sweat channel, vertical section throughout its entire course through epidermis. (Author's collection.)

The epidermis varies in thickness from about .25 mm. to 1.65 mm. The epidermis at birth is very delicate, ranging from .15 mm. to .25 mm. in thickness.

The epidermis may with essential accuracy be described as an exclusively epithelial tissue, for, excepting non-medullated nerve fibrils and minute elastic fibrils, both of which are found only among the lower cells of the epidermis and are an inconsiderable factor in its structure, it contains no fixed tissue besides epithelial cells. It is nourished by th lymph which, passing up from the lymph spaces of the corium, circulates in its intercellular spaces. It rests closely upon the surface of th corium, with which its cells are in direct contact. It was formerl believed that between it and the corium there was a homogeneous bas ment membrane; the existence of such a basement membrane is now debated question.

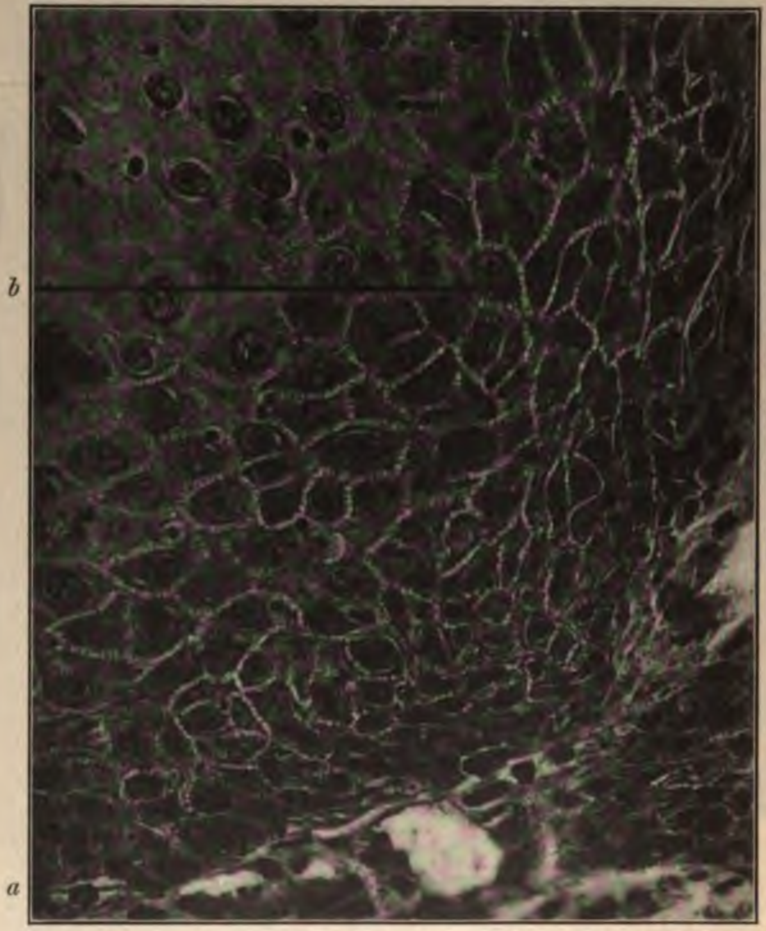

FIG. 6.—MICRO-PHOTOGRAPH OF PRICKLE CELLS OF CONDYLOMA ACUMINATUM. *a*, Basal layer; *b*, prickle cells. (Author's collection.)

The epidermis consists broadly of two layers, the mucous layer an the horny layer, which from the differences in the consistence and shap of their cells are sharply differentiated. Intermediate between these ar two layers, well called transitional layers, whose borders are not so sharpl defined, the granular layer and the clear layer or stratum lucidum. Finally, the lowest row of cells of the mucous layer is separable from the rest as the basal layer. We thus have four layers and one sublayer of the epidermis, which, enumerated from below, are as follows:

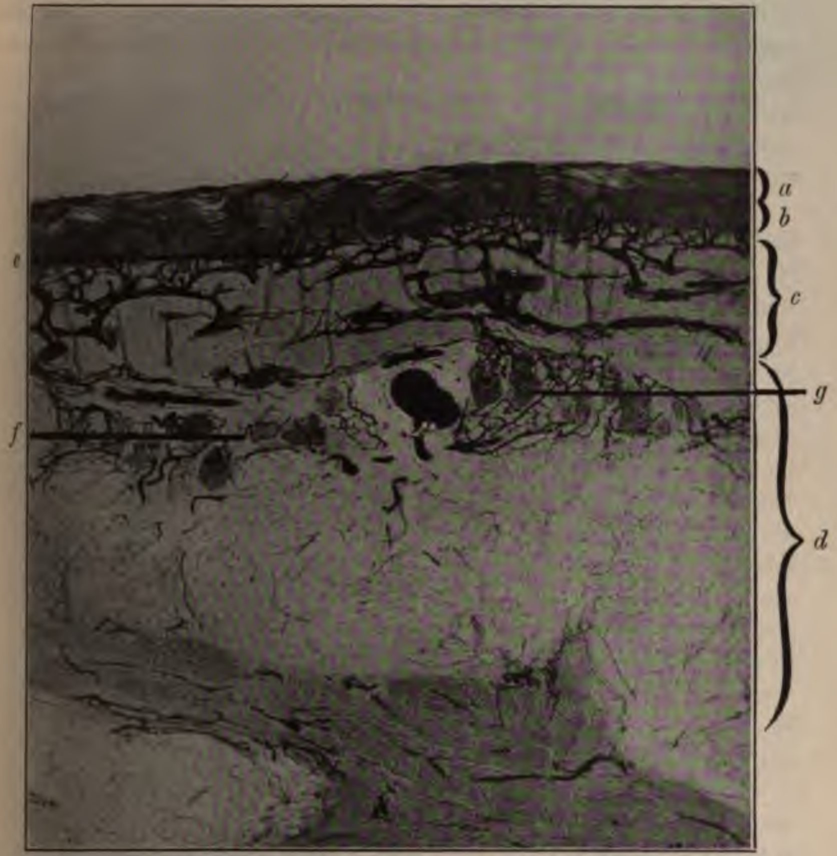

Fig. 7.—Section of Skin showing Injected Blood Vessels and Complete Vertical Section of Many Sweat Tubules and Coils. *a*, Stratum corneum; *b*, papillary layer and stratum mucosum; *c*, body of corium; *d*, subcutaneous tissue; *e*, subpapillary plexus of blood vessels; *f*, subcutaneous plexus of blood vessels; *g*, sweat coils. The blood vessels of the body of the corium and of the papillary plexus are unusually large, because having no muscular coat they dilate more fully than the vessels of the subcutaneous plexus. (Author's collection.)

(1) The stratum mucosum[1] or mucous layer, of which the lowest row of cells forms (*a*) the stratum germinativum, or basal layer.

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[1] Other names frequently given the stratum mucosum, and equally well established by usage, are rete mucosum, stratum Malpighii, stratum spinosum, prickle cell layer.

(2) The stratum granulosum, or granular layer.

(3) The stratum lucidum.

(4) The stratum corneum, or horny layer (Fig. 9).

This division is to a certain extent conventional, for the characteristics of the cell upon which the divisions are based gradually chang

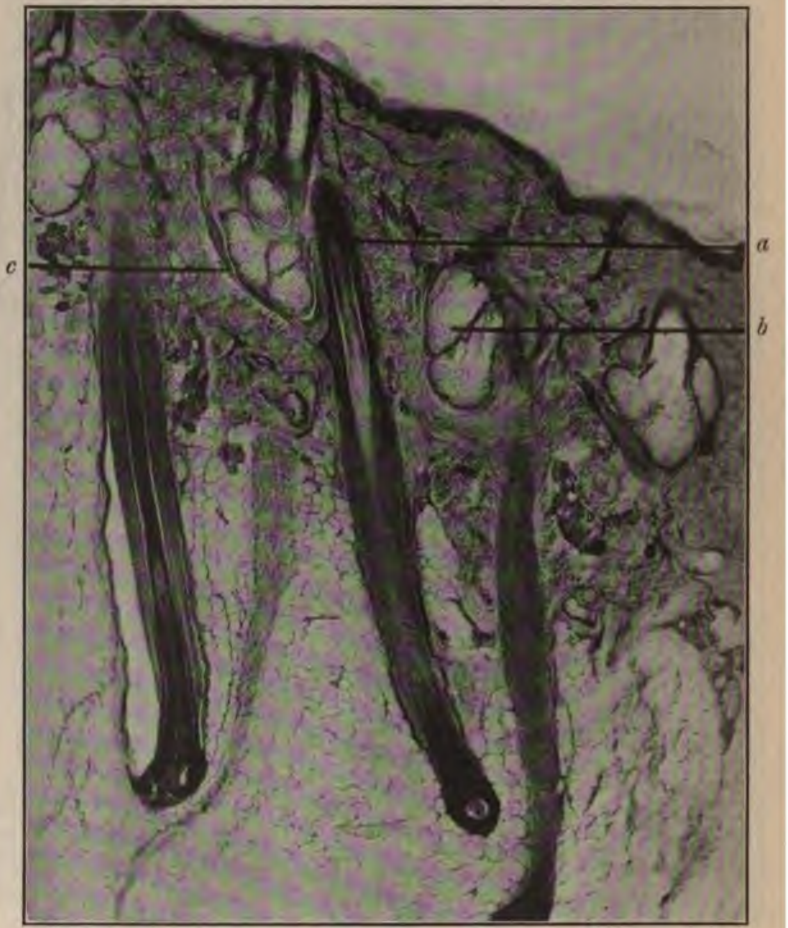

FIG. 8.—VERTICAL SECTION OF SKIN OF SCALP, SHOWING HAIRS, SEBACEOUS GLAND AND ARRECTOR PILI MUSCLE. *a*, Vertical section of hairs and hair follicles; *b*, s baceous glands; *c*, arrector pili muscles. (Author's collection.)

from the type of one layer to that of the next without abrupt transition Curiously, the one layer whose boundary is sharpest is the subdivisio the basal layer. These several layers represent the gradual change fro structurally perfect epithelial cells to structureless horny scales. Th shapes of the cells in the different layers correspond to this gradu change; in the lowest layer they are vertically columnar, then polyhedral, then roughly oval with their long diameters horizontal; then flattened so that their vertical sections are spindle shaped, then flat. The nuclei correspond in shape to that of the cells which contain them, until in the uppermost horny scales they become lost. The horny and mucous layers are present everywhere in the skin; the granular layer is absent from some points, and is not always readily demonstrable; the stratum lucidum is only present where the epidermis is thick, and is best developed on the palms and the soles. Its demonstration requires a delicate and special technique.

Basal Layer.—The basal layer is the lowest layer of cells of the mucous layer and rests upon the papillary surface of the corium. It consists usually of a single layer, but occasionally of two layers of cells. The cells are columnar or cuboidal, with oval nuclei, and are placed with their long diameters vertical to the papillary surface. The salient characteristic of this layer is that it is the germinal layer of the epidermis, and from the proliferation of its cells the epidermis is maintained. The cells divide by indirect mitosis; the daughter cells are pushed upward, and do not further divide. The epidermis is firmly attached through the basal cells to the corium. The attachment is probably dependent upon three facts: first, the cement substance which binds together the various structures of the corium probably acts as a means of attachment between the cells of the papillary surface and the basal cells; second, the cells of the basal layer are serrated on their under surfaces, and these serrations fit into similar serrations on the upper surface of the external cells of the corium (Fig. 10); third, fibrils of elastic tissue pass up from the papillary surface to spaces between the cells of the basal layer like threads to fasten the two layers together.

Mucous Layer.—The cells of the mucous layer above the basal layer are not arranged in regular rows, but are irregularly piled upon each other. Below they are roundish or polyhedral, and in the upper part of the layer they become oval or somewhat flattened. The cells of this layer have the structure of typical epithelial cells. Each cell consists of a framework of protoplasmic fibrils called spongioplasm, the meshes of which contain a homogeneous semifluid albuminous substance called hyaloplasm. The nuclei correspond in structure to the cell bodies. Each nucleus consists of a network of fibrils of chromatin, which at the periphery is condensed into a membrane that forms a nuclear wall, and in the meshes of the chromatin there is a homogeneous albuminous substance.

The peculiarity of the epithelial cells of the mucous layer is that the fibrils of spongioplasm extend out beyond the body of the cells and are continuous with fibrils of adjacent cells, so that the cells of the layer are held together in organic connection. Indeed, the layer has been compared to one large cell with many nuclei (Ranvier). It has even been suggested that the intercellular connections are necessary to the life of the cells, but Unna has disproved this by showing that the cells may still retain their vitality after their intercellular connections are ruptured by œdema.

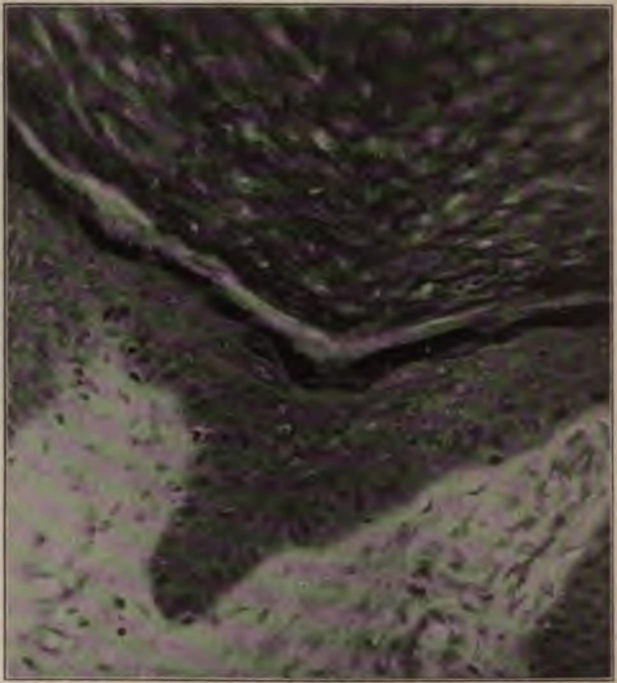

FIG. 9.—FROM A SECTION THROUGH THE SKIN OF THE HALLUX OF AN ADULT HUMAN SUBJECT. × 300. *a*, Stratum corneum; *b*, stratum lucidum; *c*, stratum granulosum, cells full of keratohyalin granules; *d*, stratum mucosum; *e*, stratum papillare corii. (Author's collection.)

These fibrils constitute the spines or prickles, from which the layer gets one of its commonest names; their character can be better grasped by remembering that they are stiff spines and not pliable fibrils. These spines firmly unite the cells together and at the same time prevent their direct contact and provide a clear space between them. It is through these intercellular spaces that the lymph circulates by which the nourishment of the epidermis is maintained (Fig. 11). The spines are demonstrable throughout the mucous layer, but are not so well developed in the basal cells as in the cells above. They are particularly prominent in conditions of abnormal proliferation of the rete cells, such as condyloma acuminatum.

Herxheimer's Spirals.—Herxheimer has called attention to "peculiar spiral fibers between the cells of the stratum Malpighii and basal layer, with occasional occurrence of small fibers between the cells of the granular and horny layers and between the cells of the inner root sheath of the hair follicles." Their nature is in question. It has been suggested that they are artefacts resulting from the method of staining; that they are elastic fibrils running up between the cells; that they are protoplasmic fibrils connecting distant cells; and that they are spirals of fibrin. The last view is the most probable one and the most generally accepted. When sections of normal or pathologically altered skin are stained with Weigert's Fibrin Stain, these deep blue colored spiral fibers can be seen in the deeper layer of the epidermis. They begin exactly at the junction of the epidermis with the derma and extend upward between epithelial cells; often they are intercellular or they may cover the cell. The reason that Jadassohn and Ehrman think that they are fibrin is that they are increased in numbers where inflammatory conditions are present in which the lymph spaces are dilated and deposits of fibrin are excessive.

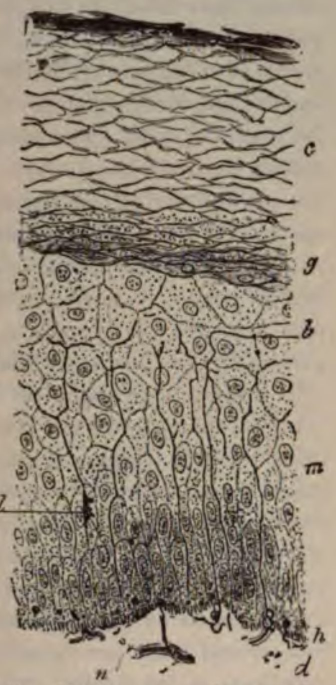

Fig. 10.—Vertical Section of the Epidermis of the Bulb of Finger of a Child Aged Fifty Days, showing Nerves and Nerve-Endings. Gold chloride preparation. *d*, corium; *m*, mucous layer; *g*, granular layer; *c*, corneous layer; *n*, nerve supplying the mucous layer; *b*, nerves and nerve-endings; *l*, Langerhans' cell; *h*, digitate under surface of basal cells. (Ranvier.)

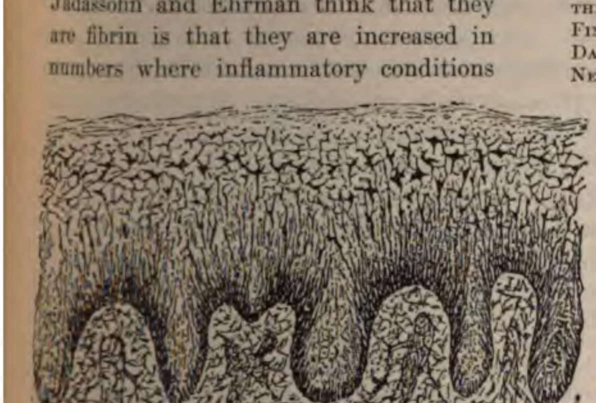

Fig. 11.—Perpendicular Section of Slightly Œdematous Skin, showing Papillary Layer and Epidermis, and Injection of the Lymphatic Spaces. (Unna.)

Granular Layer.—The granular layer consists of two or three rows of cells which represent a gradual transition from the upper cells of the mucous layer. The cells are flattened, so that they are spindle shaped on vertical section. The protoplasm is shrunken away from the nucleus of the cell, leaving a clear perinuclear space, and the nucleus is shriveled (Fig. 9). The characteristic feature of these cells is the presence of numerous granules of a peculiar substance, keratohyalin, in the body of the cells. The keratohyalin granules vary in size from minute specks to irregular lumps. They are scattered abundantly throughout the protoplasm of the granular cells, but are densest around the nuclear space, and occasional granules are found within this space. They are of importance in establishing the appearance of the skin, for, being highly refractive, they interfere with the transparency of the epidermis and with the horny cells give the skin of the white races its ground-glass-like translucence. The whitish color of young epidermis growing over wounds is produced by these granules.

STRATUM LUCIDUM.—The stratum lucidum is a narrow row of clear cells found at the under part of the horny layer on surfaces where the skin is most exposed to pressure, as the palms and the soles. It shows in sections as a translucent line, "resembling an oily streak across a sheet of paper." The cells are larger than those of the granular layer, are irregular in shape, and their nuclei are still further shriveled, or disintegrated (Fig. 9). The cells do not contain keratohyalin granules, but instead there are found droplets or puddles of a homogeneous waxy substance called eleidin. These droplets and lakes are found in the cells and between them, and their presence is the characteristic feature of the layer.

HORNY LAYER.—The horny layer is a tough, dense, insensitive, and impervious membrane. It is not easily destroyed by acids, although it is dissolved by alkalies. It does not readily absorb substances brought in contact with it, and it furnishes an effective protection against mechanical injuries and against the absorption of toxins and the invasion of microörganisms. It is thickest on the palms (.3 mm. to .4 mm.) and the soles (.5 mm. to .6 mm.) (Fig. 5), and thinnest on the face and flexor surfaces, where it is as thin as .02 mm.

It consists of cells which have undergone a greater or less degree of cornification. These are all flattened, but vary considerably in size and shape. In the deeper parts of the layer the cells are not entirely structureless. They are flattened, but not quite flat, their surface faceted from pressure of adjoining cells, and they contain a central space usually without a nucleus. Nearer the surface, where cornification becomes complete, they finally become flat and structureless horny scales. Throughout the horny layer the cells are arranged in more or less definite strata; this stratification is particularly well marked upon surfaces much exposed to pressure, like the palms and the soles, where layers of cells in different stages of cornification alternate with each other (Fig. 5). This arrangement in strata shows very well in sections, as the more completely cornified cells stain more deeply than those in which the process is not so far advanced. The intercellular processes of the epidermal cells persist until the cells exfoliate at the surface. In the process of cornification they become converted into horn substance, and thus furnish an exceedingly firm attachment between the cells of this layer.

Cornification.—The process by which the typical epithelial cells of the deeper parts of the epidermis are finally converted into horny scales on the surface has been the subject of much discussion and investigation, a large part of which has hinged upon the question as to the rôle played in this process by keratohyalin and eleidin.

Regeneration of the Skin.—The epidermis and the several epithelial appendages are regenerated by proliferation from the deeper layer of epithelial cells. In wounds and burns cells are reproduced, unless the deepest layer of the epidermis is destroyed, in which case the repair takes place from the margins. In this process of repair the glands and hair follicles send out prolongations of epithelial cells which stud the surface of the ulcer. These little islands of peculiar whitish young epithelial cells growing over wounds contain keratohyalin granules.

Keratohyalin, Eleidin, and Keratin.[1]—Keratohyalin is a semisolid or solid substance, insoluble in alcohol, ether, chloroform, weak acids, or weak alkalies. It is destroyed by strong acids and alkalies and is digested by pepsin and pancreatin. It is not stained by osmic acid or iodine, showing that it is neither fat nor glycogen. It is readily stained by many dyes. Eleidin is a homogeneous substance, probably of the consistence of fluid or semifluid fat. Like keratohyalin, it is insoluble in ether, soluble in strong acids and alkalies, and digested in pepsin and pancreatin, and does not stain with osmic acid. Unlike keratohyalin, it is slightly soluble in alcohol and it does not readily take stains. Keratohyalin and eleidin are therefore closely similar but not identical substances. It is probable that they are the same substance in different physical states and that keratohyalin is an antecedent of eleidin.

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[1] The granules characteristic of the granular layer were first studied by Kölliker, Langerhans, and Ranvier. Ranvier believed that the substance of which they were composed was fluid and gave it the name of "eleidine" from a fancied resemblance to oil. Waldeyer from a study of its chemical characteristics, and particularly from the fact that it swelled in weak alkalies, demonstrated that it was at least a semisolid. He believed that the substance bore an affinity to hyalin, and as Unna had pointed out its relationship to cornification, Waldeyer suggested the name keratohyalin. This substitution of names has been the cause of much subsequent confusion, particularly among French writers who have held to Ranvier's term. Present usage is definitely limiting the term eleidin to the waxy substance found in the stratum lucidum, while keratohyalin is applied to the granules of the granular layer.

The horn substance of the skin is called keratin. This is a homogeneous, albuminoid, solid material, chemically and physically highly resistant. It is soluble in alkalies, but it is not digested by pepsin or pancreatin, and it is affected only by strong mineral acids; it will withstand prolonged exposure to 50 per cent mineral acids. It stains deeply with most dyes. It differs, therefore, from keratohyalin and eleidin in its much more solid consistence and in that both the latter are readily destroyed by acids and are digestible in pepsin and pancreatin.

According to Unna, keratohyalin granules are found around the nuclei of the cells as far down as the middle of the mucous layer. They are abundant, however, only in the cells of the granular layer, of which they form the salient characteristic. In the stratum lucidum keratohyalin is not found, but in its stead there appears eleidin. Both are absent from the horny layer. Unna first called attention to the association of keratohyalin with the process of cornification, and, after this relation was pointed out, one view of keratohyalin was that it represented the first appearance of keratin in the gradual cornification of the epidermal cells. This view will not bear critical examination, for, as already indicated, both keratohyalin and eleidin show distinct physical and chemical differences from keratin. Moreover, various pathological conditions show that the granular layer and the horny layer may vary in their development quite independently of each other, so that the quantity of keratohyalin present may bear no direct relation to the quantity of keratin which is formed, and, indeed, cornification takes place in the entire absence of keratohyalin at the mucous junctures and in the nails.

Another view of cornification was that keratin was formed through the combination of keratohyalin and the protoplasm of the cells, thus forming keratin directly (Waldeyer), or indirectly through the formation of an intermediate substance called prokeratin (Reinke). Neither of these views is tenable in the light of Unna's digestion experiments, in which he showed, by treating horn cells with pepsin-hydrochloric-acid mixture, that part of the cell is digestible, and is not, therefore, keratin. He demonstrated by these experiments, which have been confirmed by many other observers, that in the process of cornification a shell or mantle of keratin is formed at the periphery, but that the central part of the cell does not take part in this process. The process of cornification, in short, does not consist of the gradual conversion of the entire cell into keratin, but is a metabolic process of the cell which ends in the conversion of part of the cell into keratin and of part of it into other substances. Unna's experiments render extremely probable the theory suggested by Cajal that the process of cornification consists of the gradual conversion of the spongioplasm of the cells and of the prickles into keratin, and that in this metamorphosis, which is an active process and not simply a degeneration, the rest of the cell is converted into other substances. According to this view keratohyalin and eleidin are separation products in the process of cornification, formed probably from the nucleus and the hyaloplasm of the cells, and they are ultimately converted, not into keratin, but into the fatty substance found in the cells of the horny layer which Ranvier has compared to beeswax.

Pigment.—There is very little pigment in the skin of the white races except in the hairs and in the areas which are normally dark. It appears, however, in the skin of any part which has to bear frequent exposure to light. As a rule, there is less in women and children than in men. The function of the pigment in the skin is to protect the underlying structures from the actinic effects of light, and the quantity which is present accordingly varies with the exposure of the parts to light. The ordinary increase of pigment resulting from the action of actinic energy is manifested in the familiar phenomenon of "tanning." The extreme example of this phenomenon of the production of pigment to

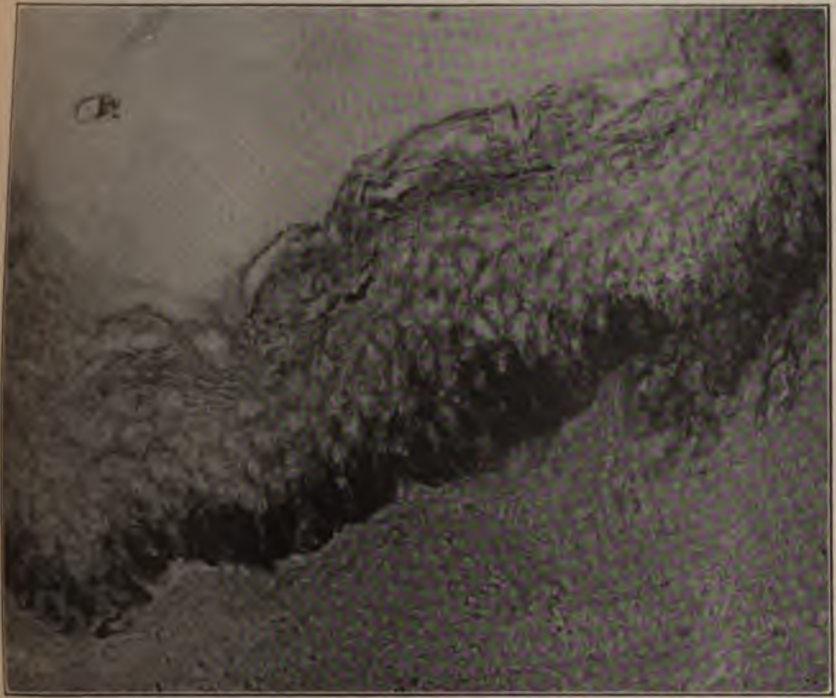

FIG. 12.—UNSTAINED SKIN OF PREPUCE OF NEGRO CHILD ONE DAY OLD, SHOWING ABUNDANCE OF PIGMENT. (Author's collection.)

protect underlying structures from the effect of light is seen in the negro race, in which the quantity of pigment in the skin renders it quite opaque. It is commonly stated that pigment does not appear in the skin except in the hairs until after birth, not even in negroes. I have examined, within a few hours after birth, ten negro babies with special reference to this point, and there can be no doubt as to the incorrectness of this statement. In no case could there be any uncertainty concerning the abundance of pigment present in the skin. In all instances the color approximated what it would be expected to be at maturity. One full-blooded negro fœtus at the eighth month which I examined showed an abundance of pigment macroscopically and upon microscopic examination, and Thomson found it in the skin of a five-months' negro fœtus. The microphotograph of a section from a negro prepuce, which was excised forty-eight hours after birth, shows the abundance of pigment present (Fig. 12).

In white races the pigment is found chiefly in the basal layer. It occurs in the form of amorphous granules in the cells and in the intercellular spaces. In the cells it is situated chiefly at the periphery, the central zone and the nucleus being free. Where it is more abundant it is found in the deeper cells of the mucous layer, as well as in the basal cells, and occasionally in the connective-tissue cells of the papillæ. In dark races it is found throughout the mucous layer and in the upper part of the corium.

The cutaneous pigment is a substance called melanin.[1] It is found also in the pigmented parts of the eye and normally or pathologically in various tissues. Melanin occurs in the form of amorphous granules which vary in color from light brown to black. It is an albuminous substance, rich in sulphur and free from iron. It is soluble in ether, acids, and alkalies, and is bleached by hydrogen peroxide and chlorine. There have been two leading views as to the origin of melanin in the skin. According to the first view melanin is produced by the breaking down of the blood pigment in the connective tissue, and the pigment so produced is taken up by leucocytes (Schmidt), or connective-tissue cells (Karg and Kölliker), or special pigment-bearing cells (Ehrmann's melanoblasts), and by these "chromatophores" is delivered to the epithelial cells. Against this view is the fact that neither in the blood nor in the neighborhood of the blood vessels are there present evidences of an escape of the red blood cells or of a disintegration and solution of them (Ziegler). Further, melanin presents characteristic differences from the hæmatogenous pigments. It differs from hæmatoidin in that it always occurs in the form of amorphous granules, while hæmatoidin occurs in the form of crystals or plates. From hæmatin and hæmosiderin it is distinguished by the fact that it is free from iron or contains it only in infinitesimal quantities, while both of these contain it abundantly. So that, even assuming that it is derived from the blood pigment, it is still necessary to invoke the assistance of cellular activity to account for its formation. The second view is that melanin is elaborated by the activity of the cells. In support of this view is the fact that its production is evidently influenced by the mechanism that controls cellular activity. This is seen by its physiological increase in certain areas in pregnancy; by its variations in certain pathological conditions, as Addison's disease, and leukoderma; by its increased production when the skin is irritated by exposure to actinic energy, as sunlight or x-rays. Especially is the rôle of the cells in its production shown in the superabundant production of pigment in pigmented moles and congenital nævi. In the cells of these is found an enormous amount of pigment, while in the cells of the directly adjacent normal skin little or no pigment will be found, although no factor to account for the difference in the quantity present can be invoked, except the special capacity for the formation of pigment of the cells of the pigmented areas. Whether the pigment is formed by connective-tissue cells or by special pigment-forming cells and from these carried to the epithelial cells, or whether it is formed by the epithelial cells themselves, may be open to doubt. Pigment is produced in connective-tissue cells, muscle cells, and ganglion cells, but in the retina, at least, it is also produced by epithelial cells, and there seems no good ground for believing that it may not be elaborated likewise by the epithelial cells of the epidermis. That such is the case would seem to be highly probable in the light of all the facts.

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[1] J. Loeb on Melanin Formation, *Jour. A. M. A.*, July 23, 1904.—Ehrmann and Oppenheim, Archives LXV, p. 323, 1903.

Corium.—"The corium,"[1] in Unna's words, "constitutes the real foundation of the skin." It might, perhaps, be better characterized as the bed of the skin. The epidermis is the parenchyma, or proper structure; the corium the supporting structure. Its closely woven framework gives it the body needed to fulfill this function, and at the same time provides the strength, firmness, and toughness necessary to its purposes as a restraining and protective envelope for soft underlying structures.

The corium (Fig. 7) consists of three layers, which, enumerated from below, are as follows:

(1) The subcutaneous connective tissue, or panniculus adiposus.
(2) The reticular layer, or pars reticularis.
(3) The papillary layer, or pars papillaris.

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[1] Cutis, derma, or true skin.

As in the case of the divisions of the epidermis, this division is more or less arbitrary, as the layers gradually merge into each other. The subcutaneous connective tissue, from the large amount of fat contained in it, differs distinctly in appearance from the rest of the corium, and for that reason it was formerly, and still is frequently, described as a separate part of the skin, and not as a layer of the corium. Embryonically, however, it is essentially a part of the corium, and logically should be included with it.

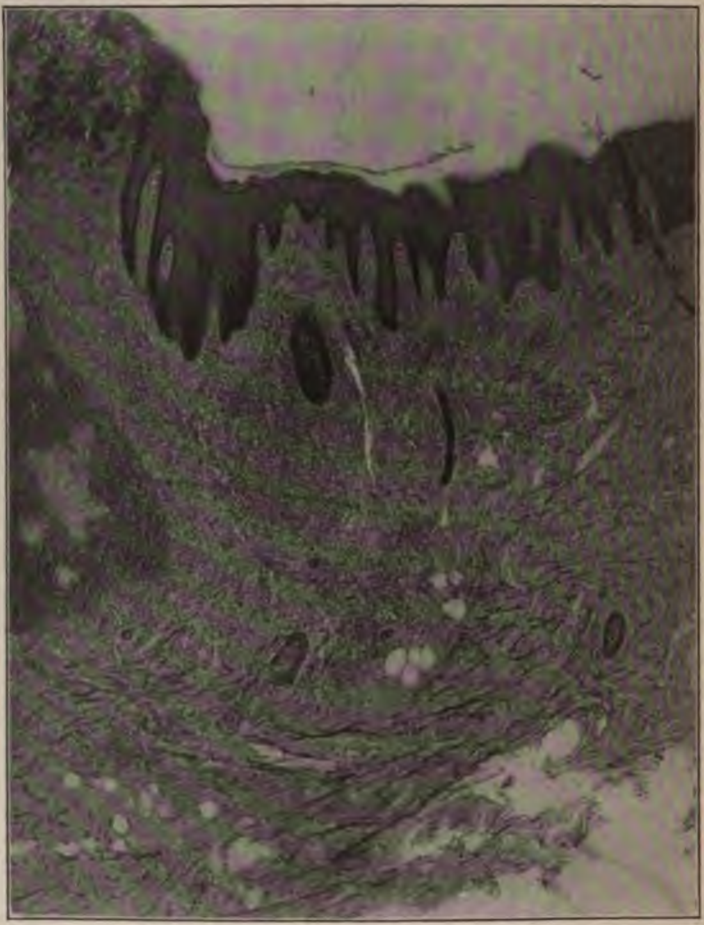


FIG. 13.—MICRO-PHOTOGRAPH OF BORDER OF NODULE OF LUPUS, SHOWING ELASTIC FIBERS (BLACK LINES); WEIGERT'S STAIN. (Author's collection.)

The corium is a connective-tissue structure, and in addition to white fibrous and yellow elastic tissue, contains all of the elements usually found in connection with connective tissue—unstriated and, in the face, striated muscle fibers, nerves, fat, blood vessels, and lymphatics. It consists of a framework of interlacing connective-tissue bundles which support and bind together the other tissues in it. The predominating tissue in this reticular framework is white fibrous or collagenous tissue, but intertwining everywhere with this are fibers of elastic tissue. The white fibrous tissue occurs in bundles which intersect in all directions to form a firm framework. This is densest in the reticular layer; it is somewhat less dense in the papillary layer; and in the subcutaneous connective tissue it forms only a very loose stroma.

The elastic fibers bind together in a fine network all of the other structures of the corium. They envelop the bundles of fibrous tissue in a network so close that at times it appears almost like a membrane. They surround the sebaceous glands, the hair follicles, the sweat coils and their tubules, the nerves and their terminals, and the blood vessels, and, passing upward from the papillary layer, fine elastic fibrils terminate between the cells of the basal layer of the epidermis. The elastic tissue may be roughly compared to a very close network of fine branching and anastomosing threads which envelop and bind together all of the other tissues. The prolongation of the terminal fibers of this tissue between the cells of the basal layer probably constitutes an important factor in binding the epidermis to the corium. The elastic fibers correspond in quantity in the different parts of the skin with the collagenous fibers. They are most numerous in the reticular layer, slightly less so in the papillary layer, and, running with the fibrous-tissue bundles around the fat lobules, they form only a loose network in the subcutaneous connective tissue (Fig. 13).

The interlacing connective-tissue bundles of the corium form roughly diamond-shaped meshes, whose long diameters are arranged parallel to the surface of the skin and correspond in direction at any given point with the line of greatest tension. It is this arrangement of the connective-tissue framework of the skin which accounts for its lines of cleavage.

WHITE FIBROUS TISSUE.—The white fibrous or collagenous tissue consists of an albuminoid substance called collagen, which is probably the anhydrid of gelatin, and is converted into gelatin by boiling. It is insoluble in water, alcohol, and salt solution, but is readily destroyed by strong acids and alkalies. The white fibrous-tissue bundles are made up of fine fibers held together by a semifluid substance similar to mucin. The fibers themselves, which are about the diameter of a red blood corpuscle, $\frac{1}{3200}$ part of an inch, are composed of fibrils whose diameters range from $\frac{1}{50000}$ to $\frac{1}{20000}$ part of an inch. The fibrous bundles normally have a wavy outline, which disappears when the skin is stretched, and on cross section are oval or round. They do not branch and anastomose, and thus do not form a true network, but fibrils frequently separate from the bundles, so that the appearance of a network is simulated (Fig. 14).

ELASTIC TISSUE.—The elastic fibers consist of an albuminoid substance called elastin. Its chemical characteristics are similar to collagen, although it is more resistant. The elastic fibers are homogeneous, straight or wavy threads which are angular on cross section. They may branch and anastomose so as to form a true network. They vary from immeasurably fine fibrils to fibers 11 microns in diameter (Stöhr). The coarser fibers are united by fine fibrils. Contrary to their name, the elastic fibers have little elasticity; are, in fact, considerably less elastic than the collagenous fibers (MacLeod), and under moderate stretching may break and their ends curl up. Their function in the corium is not to give elasticity to the skin, but rather to bind together its constituent elements.

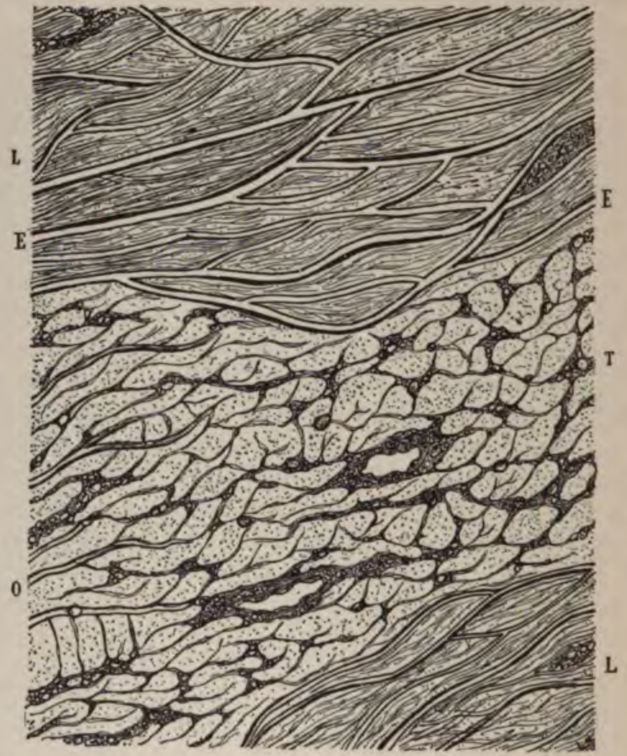


FIG. 14.—DIAGRAM OF THE MINUTE STRUCTURE OF THE CORIUM, SHOWING COLLAGENOUS AND ELASTIC FIBERS. *L, L*, Longitudinal bundles of connective tissue; *E, E*, elastic fibers bordering the bundles; *T*, transverse bundles; *O*, oblique bundles. (L. Heitzmann, Morrow's System.)

Papillary Layer.—The corium terminates upon its upper surface in the papillary layer, between which and the epidermis the line of demarcation is sharp and abrupt. The papillary contour of the upper surface of the corium presents several advantages. First, by such an arrangement certain terminals of the nerves of touch are brought nearer the surface than could be done without reducing the thickness and the efficiency of the epidermis if the upper surface of the corium were plain. In the same way the cutaneous capillaries are brought nearer the surface, an arrangement which, as will be seen, has an important bearing upon the regulation of the body temperature. Further, this papillary surface provides for a structure stronger and firmer mechanically than would be furnished by an equal amount of tissue arranged under a plain surface. And again, it furnishes a surface better adapted for the firm attachment of the epidermis.

The papillæ are of two kinds: vascular papillæ, which contain a vascular loop and are the more numerous, and sensory or tactile papillæ, in which are found terminal organs of touch (Fig. 9). The papillæ are conical or blunt projections arranged in more or less parallel ridges, on which there are sometimes one row of papillæ and sometimes two. The papillæ usually arise from a single base, but occasionally they are compound, two or more papillæ being situated upon a common base. They vary considerably in size, shape, and number in different parts of the body, and, according to Unna, become flattened in old age. They are usually about twice as long as they are broad. Over the body generally they are about .05 mm. in height. They are fewest and smallest (.02 mm.) where the skin is thin, as on flexor surfaces, and they may even be absent. They are most numerous on the penis, the clitoris, the labia minora, and the areolæ of the nipples. They are largest on the palmar and plantar surfaces, especially on the tips of the fingers and toes, and on the nipples and corona glandis (.2 mm. to .5 mm.). Sappey estimates their number at about one hundred and fifty million, a number that is probably not more inaccurate than it is incomprehensible.

Subcutaneous Tissue (*Tela subcutanea, Panniculus adiposus*).—The subcutaneous connective tissue consists of a very loose network of connective tissue whose meshes are filled with fat. The subcutaneous tissue is the fat layer of the skin, and, from the quantity of the fat in it, it is distinct in appearance from the overlying part of the corium, although there is no abrupt line between it and the reticular layer above, and, as already seen, it is essentially a part of the same structure. As it is the part for the accumulation of fat in the skin, it, of course, varies enormously in its development in different individuals and in different parts of the body. It is highly developed at birth, and its tendency to atrophy in old age accounts largely for the exaggeration of the furrows of the skin at that time of life. It serves numerous useful purposes. It gives roundness and fullness to the outline of the figure. The fat which it contains makes it a poor conductor, so that it prevents too rapid dissipation of heat. At points where it is greatly developed it furnishes an elastic, resilient protective pad against mechanical injuries. It provides a bed for the large blood vessels, lymphatics, and nerves of the skin, as well as for the deeper hair follicles and glands, and in certain localities for the Pacinian bodies. Over points where the skin is in close contact with bones, and subject to much tension and pressure, bursæ develop in it after birth. It is in this layer also that the mammary glands are situated.

According to the demands made upon its different functions in different parts of the body, the subcutaneous connective tissue varies greatly in its development. It is thin on the palms and the soles and at the various points where it is in close contact with bony structures, as on the scalp and over the various joints. It is absent on the eyelids and penis and scrotum.

ATTACHMENT OF THE SKIN.—The attachment of the skin to the underlying structures is by means of the connective-tissue network of the subcutaneous tissue, which is continuous with the underlying fasciæ and other fibrous structures. Where the skin is freely movable the connective-tissue bundles pass down obliquely through the subcutaneous connective tissue and are thus made longer, and so provide for greater motion of the skin than would otherwise be possible. Where the skin is firmly bound down these bundles are short and numerous and tend toward a vertical direction. These areas of firm attachment of the skin serve an important purpose in providing fixed points which place a limit upon the motion of the more freely movable skin stretched between them. The most important of these fixed points are the scalp, the orifices of the body, the palms and the soles, and most of the joints and bony prominences.

FAT.—The fat of the subcutaneous connective tissue is of the ordinary type found in loose connective-tissue structures in various parts of the body. It is arranged in lobes, which are surrounded by connective-tissue capsules called trabeculæ. These lobes are made up of lobules which are themselves composed of large roundish fat cells. Between the lobules there is a similar but more delicate connective-tissue capsule, and this division extends even to the fat cells which are inclosed by fine connective-tissue fibrils. The fat lobes are abundantly supplied with blood vessels and lymphatics. An arterial unit passes to each lobule, and this divides into capillaries which surround the individual cells. These capillaries in turn connect with venules that have a similar distribution. Lymph spaces occur between the fat cells and lobules, and connect with interlobar lymphatic vessels of distribution similar to that of the blood vessels. In addition to the ordinary lobules, Warren has described, particularly in situations where the reticular layer is thick, fat columns which pass up from the subcutaneous tissue to the ends of the hair follicles and sweat glands.

The fat in the subcutaneous connective tissue, like that in other connective tissue, consists of large oval or polyhedral cells which have a thin homogeneous wall, at the side of which can usually be detected a flattened nucleus. The fat first appears in the connective-tissue cells as minute droplets; these coalesce into drops, until the entire body of the cell is filled with fat and the nucleus pushed against the cell membrane. It is likely, as is generally held, that the development of fat can occur in any fixed connective-tissue cells, although certain authorities maintain that the production of fat in connective tissue is a function of special fat-producing cells.

In addition to its occurrence in the subcutaneous tissue, fat is also found in the lymphatics of the corium, in the epithelium and lumina of the sweat glands, in the sebaceous glands, and in the cells of the stratum corneum.

ORIGIN OF CUTANEOUS FAT.—Krause demonstrated many years ago that fat was secreted by the sweat glands, and in recent years Unna has built up a theory of the formation of subcutaneous fat upon this fact. According to his view fat is secreted by the sweat coils, and the globules thus produced are taken up by the lymphatics and are then filtered out from them into the connective-tissue cells around the blood vessels. This assumes an entirely different method of formation of fat in the connective tissue of the skin from that occurring in connective tissue elsewhere, where there are no coil glands to secrete it. The collection of fat in connective-tissue cells is a normal process in areolar connective tissue in all parts of the body. It is a direct and, there is no reason to doubt, an active process of the connective-tissue cells, and there seems every reason to believe that this capacity of connective-tissue cells generally accounts for the formation and deposition of fat in the subcutaneous connective tissue.

The fat found in the cells of the horny layer and in the sebaceous glands is not a derivative, like the rest of the fat in the skin, from connective tissue. As has been seen, the fat in the horn cells of the epidermis is a by product, as it were, in the conversion of the cells into keratin. The fat of the sebaceous gland is a similar product of the metabolism of the epidermal cells, but here the fat-producing function of these cells is specialized, with the result that the formation of fat is the chief product and the keratin-forming function of the cell is abortive. The fat of the sebaceous glands results from the elaboration of very fine drops of fat in the protoplasm of the cells of the lobes of the glands; these fine drops increase in size and run together, and, while this is going on, the rest of the cell is reduced to débris, which is eliminated with the fat.

Blood Vessels.—The blood supply of the skin is abundant everywhere, but is greatest on the palms, soles, and face, where the vessels are unusually large. It is more abundant on the flexor than on the extensor surfaces of the limbs. It is generally abundant upon surfaces exposed to pressure. The branching of the blood vessels is greatest where the skin is most movable (Spalteholz).

The vascular network of the skin (Fig. 7) is described schematically and with practical accuracy as distributing itself from two sets of blood vessels, the deep or subcutaneous plexus and the subpapillary plexus. The arteries passing up to form the cutaneous network divide into a deep plexus at about the juncture of the subcutaneous tissue and the reticular layer. From this plexus vessels are given off to the fat lobes, to the coils of the sweat glands, to the deeper sebaceous glands, and to the hair follicles, and other branches pass up through the corium to the subpapillary plexus. The interlobar arteries of the subcutaneous tissue give off branches to the lobules, and these again divide into capillaries and surround the fat cells. Other branches are given off from the interlobar arteries to the roots of the hair follicles and to the coils of the sweat glands. From these capillaries the venules unite to form veins whose distribution follows that of the arteries. From the deep plexus the arteries pass up more or less vertically through the corium, some of them following the course of the sweat glands and hair follicles, and others passing up independently through the areolar tissue. The general direction of these vessels is vertical, but where the tension of the skin and its movement is great their course is oblique. Just below the papillary layer the second plexus is formed, from which capillaries are given off upward to the papillæ and downward to the upper part of the tubules of the sweat glands and to the hair follicles and the sebaceous glands. The returning veins, following a similar distribution, are collected in a subpapillary plexus, pass down through the corium, and unite with the veins of the deeper part of the corium to form a deep plexus.

The arteries of the subcutaneous plexus contain a muscular coat and an outer connective-tissue coat, but above the middle of the corium the outer coats disappear and only an endothelial wall is left. In the venules passing down from the corium the muscular coat and the adventitia appear at the lowest part of the corium, and valves first occur in the veins of the subcutaneous tissue. The blood vessels of the external half of the corium are thus really capillaries without the vasomotor control for which the muscular coat is necessary. The state of their distention, therefore, is passive, being dependent, on the one hand, upon the pressure of the surrounding tissue, and, on the other, upon the pressure of the blood which reaches them.

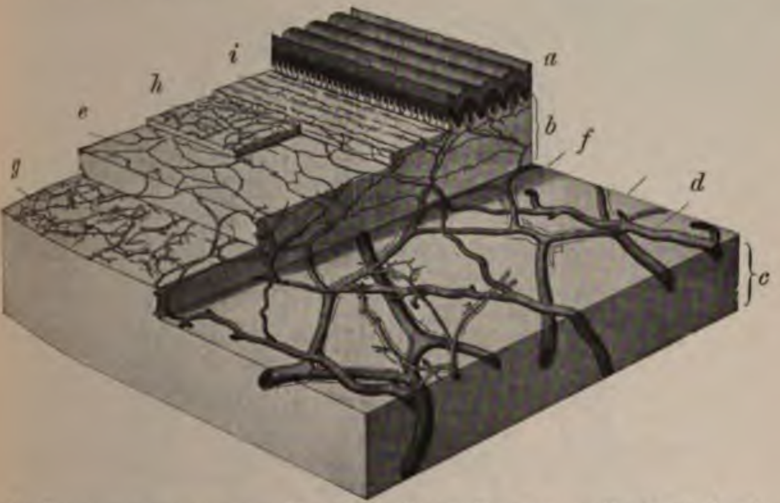


FIG. 15.—RECONSTRUCTION OF THE CUTANEOUS BLOOD VESSELS. *a*, Epidermis; *b*, subcutaneous tissue; *d*, deep, and *e*, superficial arterial plexus; *f–i*, successive venous plexuses. × 9½. (Spalteholz.)

Lymphatics.—The corium and the mucous layer of the epidermis are bathed in lymph; but the circulation of the lymph in the skin is chiefly in unlined lymph spaces, and true lymphatics with a definite endothelial lining are relatively few. The uppermost lymphatic vessels begin as blind loops in the lower part of the papillæ, and after forming a subpapillary plexus pass down obliquely through the corium to a plexus of interlobar lymphatics in the subcutaneous tissue. Their distribution thus follows that of the blood vessels. Numerous small lymphatics connect in all parts of the skin with the lymph spaces. The lymph spaces are interstices between the cells and fibrils of the skin. Through these the lymph is supplied abundantly to all the tissues of the corium, and from these spaces in the papillary layer the lymph passes up and circulates in the interepithelial spaces of the epidermis. According to Key and Retzius the lymph passes into the epidermis from the apices of the papillæ and returns to the corium through the interpapillary depressions. According to Klein the lymph passes from the lymph spaces into the lymphatics either between the endothelial cells or through stomata in their walls, but that the communication is continuous between the lymphatic vessels and the lymph spaces is shown by the fact

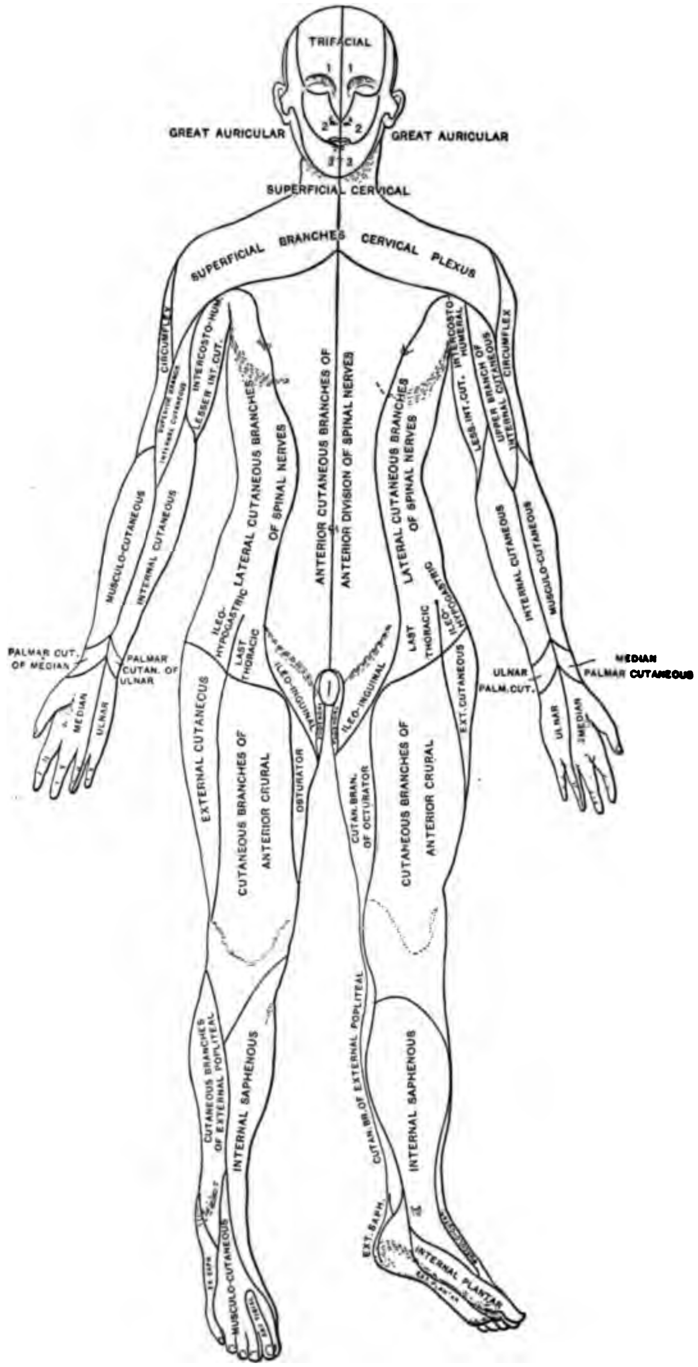
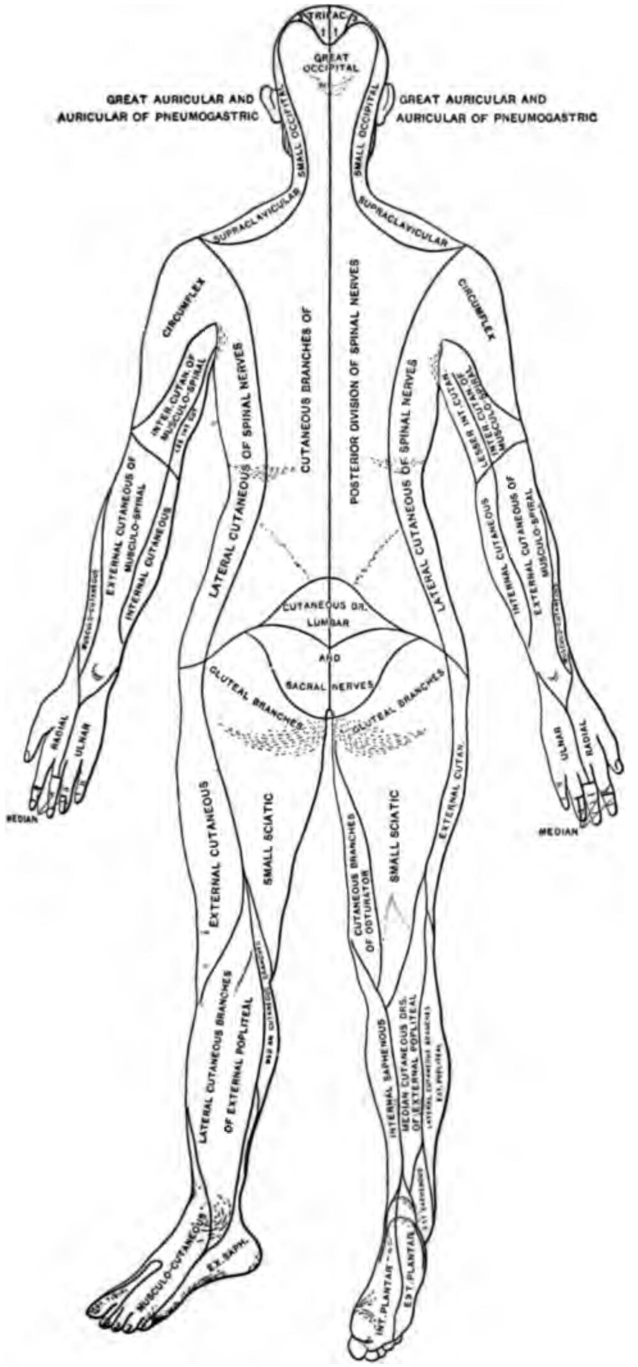


FIG. 16.—NERVE SUPPLY TO THE SKIN. Anterior Aspect. (Duhring.)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EXT. PLANTAR

Fig. 16*a*.—**Nerve Supply to the Skin.** Posterior Aspect. (Duhring.)

that the spaces can be injected from the lymphatic vessels of the subcutaneous tissue (Fig. 11).

Muscles.—The skin contains involuntary muscle fibers and, in the face, voluntary muscle fibers. The involuntary muscles of the skin are: (1) the arrectores pilorum (Fig. 8), which are widely distributed in connection with the hairs, and (2) layers of muscle fibers found in a few places. In addition, there have been described diagonal muscle fibers in the forehead, cheeks, and back, which are placed obliquely in the corium and are analogous to the arrectores pilorum, but are not inserted in hair follicles. Unna has placed these, very naturally, it would seem, in one class with the arrectores pilorum as the oblique tensors of the skin. The arrectores pilorum are inclosed in a fibrous sheath, through which they arise from the connective-tissue network of the papillary layer and are inserted in the connective-tissue sheath of the hair follicles. They are inserted into the hair follicles on the side where the latter form an obtuse angle with the skin and pass around the sebaceous glands. When they contract, therefore, there occurs, in addition to a puckering of the skin at their point of origin, more or less erection of the hair (cutis anserina). This condition may be excited by various forms of stimulation of the skin, as cold, exposure to light, stroking the skin. The sensitiveness to the reaction varies in different individuals. Maxwell has reported a case in which the individual could voluntarily produce cutis anserina. It was formerly held that the contraction of these muscles caused expulsion of the sebum, but this is now not generally admitted. The arrector muscles are most highly developed in the scalp, and are abundant in the axillæ, lips, and eyebrows; they are rudimentary with the lanugo hairs (Köllicker).

Nonstriated muscle fibers arranged in horizontal layers exist in the dartos of the scrotum, areolæ of the nipples, and the eyelids. In general the bundles are arranged transversely to the lines of cleavage of the skin; in the scrotum the bundles are parallel to the median raphe; in the eyelids they radiate to the edge of the lid; and they occur in concentric circles in the nipples and their areolæ. By their contraction the skin is thrown into folds. The striated muscles of the skin are muscle fibers which represent the insertion of the muscles of expression of the face in the corium. Similar fibers also come from the platysma. Their contraction, along with the contraction of the larger muscles to which they belong, causes the wrinkling which is shown in the expression of the emotions.

Nerve Supply of the Skin.—The skin is universally supplied with sensory nerves. In addition there are motor nerves which pass to the muscular tissue of the skin, vasomotor nerves in connection with the muscular coats of the blood vessels, and possibly, but not probably, independent trophic fibers which control the tissue changes and nutrition of the skin. The distribution of the nerve supply of the skin is shown in the accompanying figures, taken from Duhring (Figs. 16 and 16*a*).

The cutaneous nerve supply is most abundant in the palms and the soles, especially in the tips of the fingers and the toes, where the sensation of touch is most acute.

The nerves of the skin consist of both medullated and nonmedullated fibers. The distribution of the nerves in the skin follows the same course as the blood vessels. The larger trunks in the subcutaneous tissue divide into fibers, which give off branches to the surrounding tissues and pass up with the blood vessel through the corium to form an abundant horizontal plexus beneath the papillæ In their course through the corium they give off branches to the hair follicles, sweat glands, and sebaceous glands, to the blood vessels, to the arrectores pilorum, and to the connective-tissue bundles. The subepithelial plexus consists chiefly of fibers which have lost their medullary sheath, and from this plexus fibrils are given off to the surrounding tissues, including the lower cells of the epidermis. Some of the fibrils of this plexus retain their medullary sheath and terminate in the touch corpuscles in the papillæ. The nonmedullated fibrils which extend into the mucous layer of the epi-

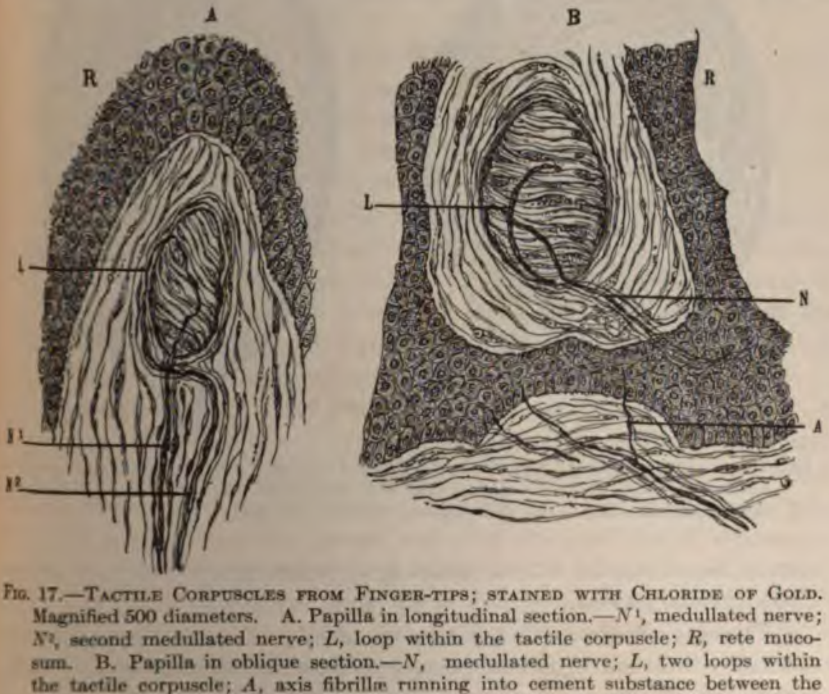

FIG. 17.—TACTILE CORPUSCLES FROM FINGER-TIPS; STAINED WITH CHLORIDE OF GOLD. Magnified 500 diameters. A. Papilla in longitudinal section.—N^1, medullated nerve; N^2, second medullated nerve; *L*, loop within the tactile corpuscle; *R*, rete mucosum. B. Papilla in oblique section.—*N*, medullated nerve; *L*, two loops within the tactile corpuscle; *A*, axis fibrillæ running into cement substance between the epithelia; *R*, rete mucosum. (L. Heitzmann, Morrow's System.)

dermis pass up for several layers of cells and either end between the cells or, as Unna holds, enter the cells and end in swellings applied to the nuclear membrane (Fig. 10). The fibrils which supply the hair follicles pass in a similar way into the external root sheath of the follicles and end similarly, either between the epithelial cells or in them. The fibrils supplying the blood vessels pass into the endothelium. In addition to the medullated fibers which terminate in the touch corpuscles, other medullated fibers end in special terminal corpuscles, the touch cells of Merkel and the Pacinian bodies.

The touch corpuscles, Fig. 17 (corpuscles of Meissner or Wagner), are found placed vertically in the sensory papillæ and are universally distributed in the skin, but are most abundant upon the palms and the soles, especially upon the tips of the fingers and the toes. They are oval or spindle-shaped bodies, composed chiefly of numerous layers of connective tissue. The nerve fiber on entering the corpuscle loses its medullary sheath, and the axis cylinder splits into fibrils which ramify between the layers and end in small swellings. Usually one corpuscle nearly fills a papilla, but occasionally there are several small corpuscles found in one papilla.

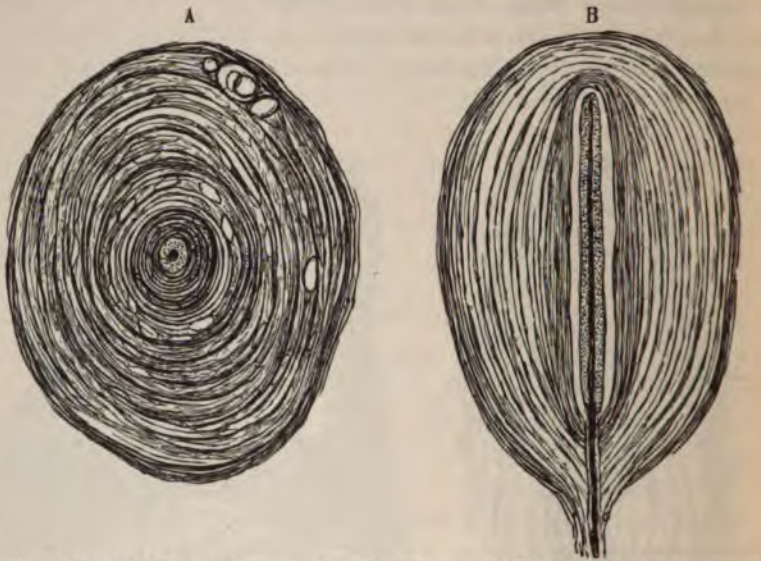

FIG. 18.—PACINIAN CORPUSCLES FROM THE DERMA OF THE PALM OF THE HAND; STAINED WITH CHLORIDE OF GOLD. Magnified 500 diameters. A. Transverse section. B. Longitudinal section. (L. Heitzmann, Morrow's System.)

Pacinian Bodies.—The Pacinian bodies, Fig. 18 (corpuscles of Vater), are found in the subcutaneous tissue, where they appear to the naked eye in unstained sections as fine, oval, milky specks. They are oval bodies, 1 mm. to 4 mm. long, and consist of a capsule which is composed of numerous superimposed layers of connective-tissue fibers which are continuous with the sheath of Schwann of the nerve. Between the lamellæ are spaces which are filled with lymph and in which an occasional capillary is found. The axis cylinder of the nerve passes into the core of the body, and beyond the center of the core splits into fibrils, which end in pear-shaped swellings. Occasionally a nerve passes through one corpuscle to end in a second. The Pacinian bodies are found in the skin only in the subcutaneous tissue, and are most abundant in the fingers and the toes. They are also found in other structures of the body. They are believed to be sensory organs for the appreciation of pressure and tension, which affect them through the lymph of the cortex acting by hydrostatic pressure on the core.

Touch Cells of Merkel.—Merkel, Ranvier, and others have described small, round- and pear-shaped cells in the papillæ which are connected with the axis cylinder of nerve fibrils. Their character and functions are not established.

Langerhans' Cells.—Peculiar star-shaped or radiating cells, described by Langerhans, are found between the epithelial cells of the deeper part of the mucous layer. They are not, as was first thought, connected with nerves, and their character is uncertain. They may be leucocytes filled with pigment.

Cellular Elements of the Skin.—The corium, like all other areolar connective tissue, contains, in addition to fibrous elements, cellular elements. The fibrous elements are the white fibers and the yellow elastic fibers which we have considered. The cellular elements are of two kinds: (1) fixed cells and (2) migratory or wandering cells.

Fixed Cells.—The fixed cells are: (*a*) connective-tissue cells proper, (*b*) mast cells, (*c*) vacuolated cells. These are all varieties of connective-tissue cells. They are all flat nucleated cells, with a greater or less amount of protoplasm around the nucleus, and they present the same variations in size and shape. They are so called because they are not ordinarily capable of independent amœboid movement.

Connective-Tissue Cells Proper.—The connective-tissue cells vary in shape and size, but are of two general types: one a cell with a large mass of protoplasm around the nucleus, the other a small cell which shows simply as a nucleus or as a nucleus with a thin border of protoplasm around it. The mature and most common type of connective-tissue cell is the large cell. This is flat and usually polygonal in outline, but frequently it has long, protoplasmic processes and is spindle shaped or stellate. In pigmented parts of the body the protoplasm of some of these cells contains pigment granules, forming the so-called pigment cells of the corium, which are not especially characteristic of the human skin, but are salient features in the corium of certain amphibians. The small form of connective-tissue cells consists of a nucleus only or of a nucleus and a thin border of protoplasm. They are somewhat larger than mononuclear leucocytes, but their resemblance to them has been the source of great confusion. These round cells are young forms of connective-tissue cells, and are found usually only in young tissue. They constitute the fibroblasts. They may develop into large connective-tissue cells. Probably the connective-tissue cells develop into the connective-tissue fibers.

Mast Cells.—Mast cells correspond in shape of nucleus and body to the connective-tissue cells proper, and their peculiar characteristic is that they have numerous coarse granules in their hyaloplasm which stain deeply with basic aniline dyes, making them prominent objects in the microscopic field. They are always present in the corium, especially around the glands and the blood vessels, but are not numerous, not more than one or two in one field with a medium power (MacLeod). They are most numerous in pigmented areas, and for that reason a relationship to pigment formation has been suggested. Their number is greatly increased in certain pathological conditions. There is much uncertainty as to their significance. It is probable that they represent a degenerating or maturing connective-tissue cell in which their granules have separated from the protoplasm. If this is true, they present an analogy to maturing epidermal cells in which keratohyalin occurs as a similar separation product. The mast cells of connective tissue are probably formed from connective-tissue cells. Similar cells, which are a form of leucocytes, are found in the blood (Ehrlich's mast cells).

Vacuolated Cells.—The vacuolated cells of Schäfer are rounded or spindle-shaped cells without processes, which take their name from their coarse network of spongioplasm, the interstices of which look like vacuoles. Schäfer believes that these are really vacuoles which contain a fluid like lymph. Occasionally mitotic figures are found in these cells, and it is possible that they are the germinal cells among the connective-tissue cells, performing a proliferating function analogous to that of the basal cells of the epidermis (MacLeod).

Appendages of the Skin.—The appendages of the skin are the sebaceous glands, the sweat glands, the hair, and the nails, all of which are developed from the epidermis and present specialized variations upon its ordinary structure.

Sebaceous Glands.—The sebaceous glands may be regarded as invaginations of almost the entire thickness of the epidermis. In their simplest form they are pear-shaped structures, but in their ordinary well-developed form are racemose glands consisting of several lobules uniting in a common neck (Fig. 8). In their most elaborate form they may contain twenty or more acini. Each gland is surrounded by a connective-tissue sheath abundantly supplied with blood vessels, nerves, and lymphatics. The gland itself has a basal layer of cuboidal or cylindrical proliferating epithelial cells continuous with the basal layer of the epidermis. Above this layer are found large cuboidal cells which show fatty changes as they approach the center. Minute fat droplets appear in the hyaloplasm; these increase in number and coalesce, until finally they fill the cavity of the cell. At the same time the spongioplasm becomes more resistant to acids, alkalies, and digestive ferments, and thus shows an abortive attempt at keratinization, although true keratin does not develop. At the center of the gland the cells break down and the fat coalesces into a mass, which, mixed with epithelial débris, water, and various organic products, constitutes the sebum.

Sebaceous glands vary greatly in their development and activity in different individuals. They are found universally over the body, except upon the palmar and the plantar surfaces and the dorsal surface of the terminal phalanges of the fingers and the toes. Their number is estimated at two or three millions. They are particularly abundant about the face and over the shoulders. They are largest on the nose, in the conchæ of the ears, around the nipples, on the penis, upon the mons veneris, the labia majora, and the scrotum. The Meibomian glands at the free border of the lids and the Tysonian glands upon the glans penis and the inner surface of the prepuce are the largest of the sebaceous glands. The mammary glands are a modified and exaggerated form of sebaceous glands.

In addition to the sebaceous glands which occur with the hairs, sebaceous glands occur independently around the nipples, upon the red border of the lips, the labia minora, and other mucous orifices, and open directly upon the surface. These have all been well classed by Unna as the sebaceous glands of the mucous orifices.

The sebaceous glands are usually found in association with the hairs. With the large hairs they appear as appendages of the hairs, and while one only is ordinarily found, occasionally two or more occur in association with a single hair. Where they are associated with the lanugo hairs they may appear as the relatively more important structure, so that the hair is rather the appendage of the sebaceous gland than the reverse. Around each gland runs an arrector pili muscle, the contraction of which probably exerts some effect upon the extrusion of the gland's contents; but there is no doubt that the expulsion of the sebaceous matter from the gland is chiefly caused by the continual formation of new sebum in the body of the gland and the pressure arising therefrom.

Sweat Glands.—The sweat glands (Figs. 5 and 7) are simple tubules, lined with cuboidal epithelium, which pass down from the epidermis into the corium and terminate in a closed end. The lower part of the tubule is rolled up into a coil which is situated either in the subcutaneous tissue or deep in the corium, and is the secretory apparatus of the gland. From the coil the tubule ascends in a winding, but upon the whole vertical, course through the corium. It always passes from the corium to the epidermis between papillæ. In the epidermis its course becomes more tortuous and forms a spiral channel. The openings of the sweat glands are usually directly upon the surface, but in certain locations, as in the axillæ and around the anus, they open into the mouths of hair follicles. The lining of the tubule in its course through the corium is epithelium continuous with that of the basal layer. In its course through the epidermis, however, it has no lining membrane, but is an open channel between the cells, so that there is direct communication between it and the intercellular spaces. In the mucous layer the cells of the epidermis are grouped concentrically

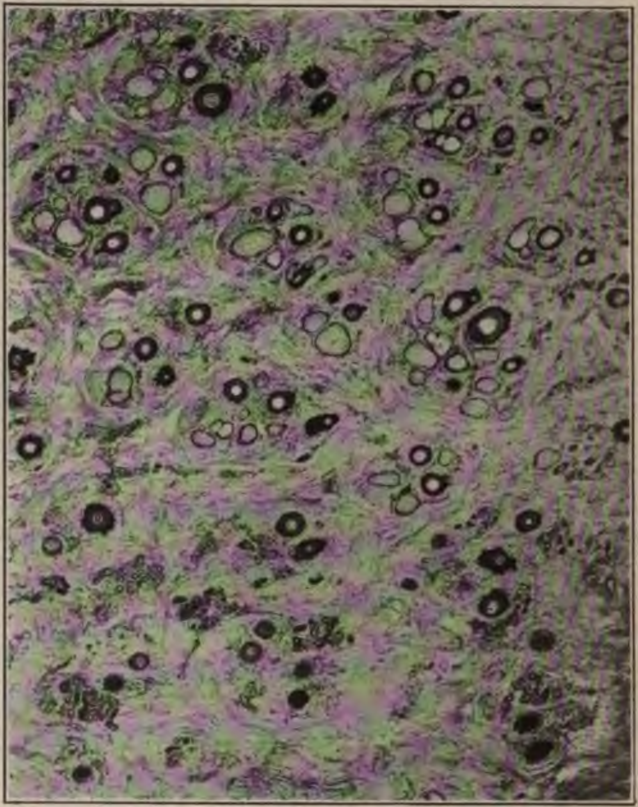

FIG. 19.—HORIZONTAL SECTION OF SKIN OF SCALP.
Showing relation of sebaceous glands to hair-roots. × 30. (Author's collection.)

around the sweat glands, and in the transitional layer the cells dip down around the sweat channel. The lumen of the tubule in the coil is greater than that of the duct and is lined with a single layer of cylindrical or polyhedral epithelial cells containing fine granular protoplasm. When the cells are actively secreting they become more granular and their nuclei are situated nearer the center than at other times (Renaut). In the cells of the coil are often found fat droplets and pigment granules. The cells of the coil are not in direct contact, and connective-tissue cells are found here and there inserted between them. Here also spindle-shaped muscle cells are found arranged spirally and longitudinally around the tubules, and also penetrating between the cells. Their function is to expel the sweat. The muscle cells are not found around the duct. The ducts of the sweat glands are lined by two or three layers of cuboïdal cells. A network of blood vessels from the deep plexus surrounds the tubules of the coil and extends up around the lower part of the duct, while other capillaries descend from the subpapillary plexus to supply the upper part of the duct. The sweat glands are abundantly supplied with nonmedullated nerve fibers, which form a fine network around the tubules. From this network fibrils pass in to end in fine bulbs on the surfaces of the gland cells.

In addition to the ordinary type of sweat gland with a single tubule, larger glands are found in the axillæ and around the anus which branch in their lower extremities. The ceruminous glands of the ear, which are large sweat glands, are of similar structure, but they differ further in that their cells are filled with fat drops and pigment granules. They approach in type the sebaceous glands.

Sweat glands are found universally over the surface of the skin, except in the inner surface of the prepuce, the glans penis, and the red border of the lips. On the palms and the soles they are arranged regularly along the papillary ridges; around the anus (glands of Gay) and around the areolæ of the nipples they are arranged in a single row in the form of a ring. In the ordinary glands the diameter of the coil averages .3 mm. to .4 mm. (v. Brunn), in the large glands it measures 1 mm. to 3 mm. (Kölliker). Their number varies greatly in different individuals. They are three and one half times as numerous on the palms and the soles (2,800 per square inch, Krause) as on other parts of the body. Their total number is estimated, like that of the sebaceous glands, at two or three millions.

Hairs.—The hairs are divided into long hairs, those of the scalp and beard; bristles, which occur in the nostrils, ears, and around the anus; and lanugo hairs, which form the down.· These do not differ in structure (Fig. 20).

In considering the minute anatomy of the hair two structures have to be studied, the hair itself and the follicle from which it grows. The two form a structure, which upon analysis furnishes an excellent illustration of the elaboration of a highly specialized structure from a more simple structure of the same type, but of quite different arrangement. Its apparent complexity is increased by a rather confusing nomenclature applied to the layers of the root sheath.

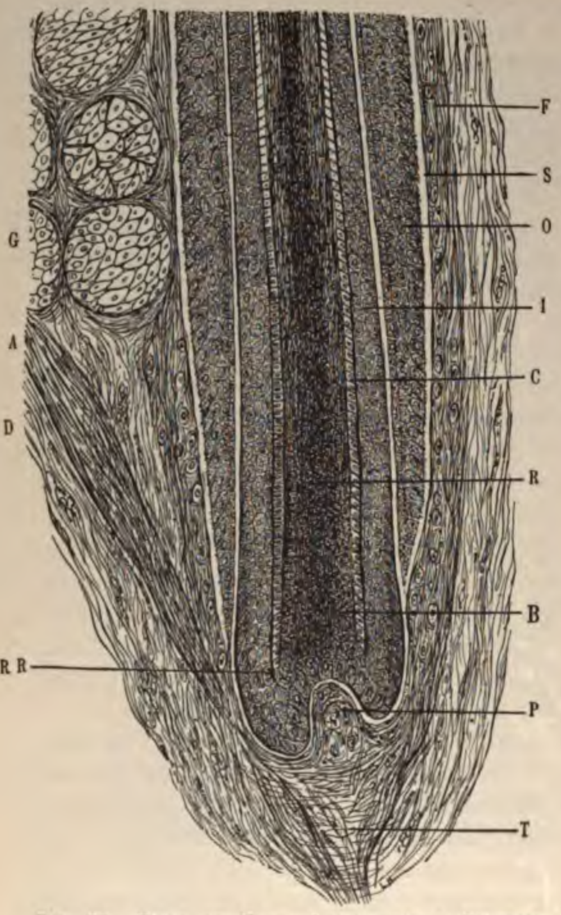

Fig. 20.—Root of Hair; Longitudinal Section. Magnified 250 diameters. *P*, Papilla of hair; *B*, bulb of root; *C*, cuticle of hair; *I*, inner root sheath; *R R*, recurvation of inner root sheath into the root; *O*, outer root sheath; *S*, facement membrane; *F*, follicle, fibrous connective tissue, holding transverse sections of smooth muscle fibers; *G*, acini of sebaceous glands; *A*, arrector pili muscle; *D*, derma; *T*, connective-tissue tract of follicle to subcutaneous fat tissue. (L. Heitzmann, Morrow's System.)

The hair may be regarded as a spine of epidermis, of relatively enormous length, the structure of which is altered and largely determined by the growth of a sheath of epidermis around it for a considerable part of its length. This sheath, the epidermal follicle, compresses the central spine of epithelium growing up through it and converts it from what might be a structure of superimposed layers of horizontal cells into a thread, the cells of which have their long diameters parallel to the axis of the hair follicle. In fulfilling its purpose as a closely enveloping sheath the hair follicle grows in two directions: the outer layer of the follicle grows toward the axis of the follicle, the inner layer of the follicle grows upward with the hair from the papilla at the base of the follicle. Thus the follicle, through the growth of its external layer, constantly exerts a lateral pressure in all directions upon the hair, forcing its cells to assume an elongated form and to arrange themselves in layers at right angles to the line of pressure—that is, vertically or parallel to the axis of the hair.

External Sheath of Follicle and Hair Papilla.—The follicle is surrounded by a dense connective-tissue sheath, called the external sheath of the follicle, which is supplied with blood vessels, nerves, and lymphatics, in the lower part from the deep plexuses, in the upper part from the subpapillary plexuses. At the base of the hair this sheath is projected upward into a papilla which is the exact analogue of the papillæ of the papillary layer. This papilla is conical or pedunculated, and the end of the hair follicle fits over it like the cup of an acorn. It is the bed from which all of the structures of the hair and the hair follicle, except the external sheath of the follicle, grow. It contains a vascular loop and medullated nerve fibers, and in its connective tissue numerous pigmented connective-tissue cells are found.

Hair Follicle.—The hair follicle (Fig. 21) is an invagination of the entire thickness of the epidermis. It extends obliquely downward through the corium and ends at the papilla, which in coarse hairs is situated in the subcutaneous tissue, in lanugo hairs in the corium. Above the opening of the sebaceous gland—that is, in its upper third—the layers of the hair follicle correspond to those of the epidermis. Below this point the follicle consists of three layers, which, enumerated from without, are: (1) the external root sheath, which would better be called the mucous or prickle-cell layer, (2) the internal root sheath, and (3) the cuticle. The external root sheath, like the mucous layer elsewhere, is composed of cuboidal prickle cells several layers in thickness, but tapering to the thickness of a single layer of cells around the neck of the papilla. The internal root sheath consists of a layer of cells which grow from the neck or base of the papilla. There the cells are polygonal, with round or oval nuclei, and contain keratohyalin granules. Above the bulb they rapidly cornify and lose their cell structure. The outer cells of the internal root sheath cornify more rapidly than the cells of the inner part of the internal root sheath, and this difference has been the basis for differentiating these cells into two layers—the outer, the sheath of Henle, the inner layer, toward the hair, the sheath of Huxley. The distinction between these two sublayers is not essential, and disappears in the upper part of the follicle, where the entire inner root sheath becomes cornified. The cuticle, situated between the inner root sheath and the hair, springs from active cells around the base of the papilla, and throughout nearly its entire length consists of a single layer of thin, transparent, elongated scales. These are imbricated and placed slanting, with their free edges projecting downward, so that they engage the corresponding cells of the cuticle of the hair—which project upward—and thus firmly anchor the hair in the follicle.

Hair.—The hair consists of a shaft and a root; the shaft is that part which projects above the surface outside the follicle, the root is that part contained within the follicle. The lower end of the root which envelops the papilla is the bulb. The structure of a complete

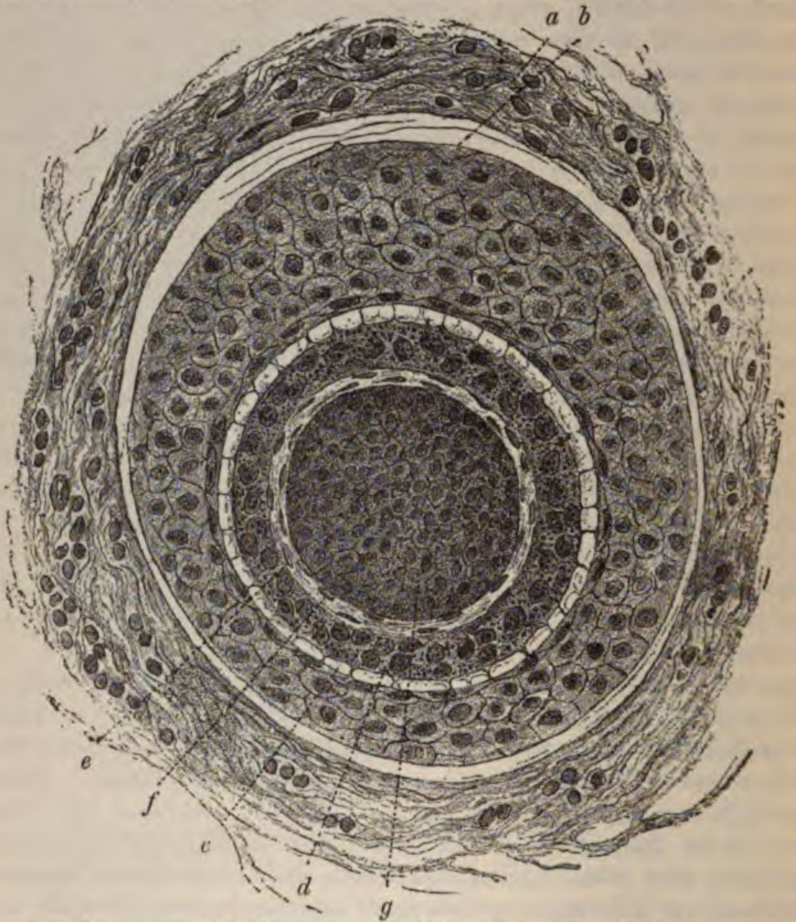

Fig. 21.—Transection of a Hair Near the Middle of the Root Sheath. *a*, Dermal root sheath; *b*, outer margin of the epidermal root sheath—the light space is the glossy membrane, the polyhedral cells form the outer root sheath; *c*, Henle's layer of the inner root sheath; *d*, Huxley's layer; *e*, cuticle of the root sheath; *f*, cuticle of the hair; *g*, cortex of the hair shaft. Highly magnified. (After Kölliker.)

hair, like that of the follicle, consists of three parts: (1) the medulla, (2) the cortex, and (3) the cuticle. In coarse hairs the medulla forms the axis of the hair and extends almost to the tip; in lanugo hairs it extends only for a short distance above the papilla. The cortex is the chief structure of the hair, and both it and the cuticle extend throughout the length of the hair. All three of these layers grow from the papilla, from active epithelial cells rich in protoplasm and with well-defined nuclei.

The medulla near the bulb is composed of cubical cells with large nuclei which contain keratohyalin granules, but no pigment. Further up the granules disappear, the cells become more flattened, and air spaces exist between them. The air spaces in the medulla have an important bearing upon the color of the hair. When they are abundant and the pigment is scanty they render the hair distinctly white, while hairs which have entirely lost their pigment but have no air spaces in the medulla are gray, and never white (Szymonowicz). The cells of the cortex in the bulb are elongated cuboidal cells, but throughout most of the length of the hair they are spindle-shaped cells with oval nuclei and lie parallel to the axis of the hair, so that they present the appearance of a fibrillated structure. In this layer pigment granules are found abundantly in and between the cells of colored hair, and near the papilla there occur branched pigment cells. There is also a coloring matter in solution which infiltrates the cortical cells. Air spaces are found also between these cells. The cortex of the hair is a firm, dense, tough structure. The cuticle, the outside layer of the hair, begins at the papilla in active epithelial cells, but throughout nearly the entire length of the hair it consists of fine, transparent, rectangular, imbricated scales. The cuticle is a layer of four to six scales in thickness, which are arranged slanting, with their free borders projecting upward. They are completely cornified.

The arrangement of the cells of the layers of the hair follicle and of the hair is thus the result, in large part, of the different sites from which they grow, of the varying conditions of pressure to which they are subjected, and, to some extent, of variations in the process of cornification in different layers, the latter variations depending upon factors which are not clear. The germinal cells for all of the layers except the external root sheath are the same. They are all cylindrical or cuboidal cells rich in protoplasm and containing distinct nuclei. The different layers, however, do not cornify at an equal rate or without variations in the process. Both the inner root sheath and the cuticle of the hair follicle show keratohyalin granules. In the hair itself keratohyalin is found only in the medulla, while neither keratohyalin nor eleidin is found in the cortex and cuticle of the hair (Szymonowicz). In the cortex and the cuticle the cells become completely keratinized.

The growth of the hair on the scalp is about one half inch per month (Duhring), but is greater in summer than in winter. It is also more rapid when the hair is cut frequently.

Shedding of the Hairs.—The hairs in man are continually shed during life, but this does not occur periodically, as it does in animals which inhabit cold climates.[1] The first shedding of hair in man is the shedding of lanugo hair, which begins between the seventh and eighth months *in utero*, and which, if not complete by birth, is completed a few months thereafter. Throughout life the shedding of the hair is normally continuous. According to v. Brunn, the life of a hair on the scalp is two to four years; according to Donders the eyelashes are changed in from three to five months, the other hairs more slowly.

When a hair is about to be shed, cornification of the root extends down almost to the papilla, the bulb splits into fibrils, and the hair separates at the papilla (Fig. 22). The lateral pressure of the follicle at the same time causes a constriction at this point. The cells of the matrix around the papilla then begin to form a new hair, and as the new hair grows up through the follicle the dead exfoliating hair is pushed upward or falls out. It must be said that, according to one view, the formation of new hair is accompanied by the formation of a new papilla, but that view seems untenable when one takes into consideration the permanent connective-tissue structure of the papilla and the analogies between it and the growth of the epidermis or of the nails from their matrices.

Hair is found upon all parts of the skin except the palms, the soles, the vermilion border of the lips, the glans penis, the inner surface of the prepuce, the labia majora, and the dorsal aspects of the distal phalanges of the fingers and the toes. They are so short upon the upper lids as not to project above the surface. Their number, size, and length vary enormously in different individuals. E. Wilson estimates about 120,000 to the scalp; v. Brunn, 300 per square centimeter on the vertex and 44 on the chin as an average. They are coarsest in the beard and about the genitals, and are smallest over the nonhairy parts, constituting the lanugo.

The normal hair tapers to a point, which may split. On cross section hairs may be circular, oval, or angular. Straight hairs are usually round; curly hairs, oval. The curl of the hair is probably further influenced by a curve in the follicle; at least the follicles of the eyebrows and lips and of the scalps of negroes are curved.

The hairs of the eyelids, cilia, of the nostrils, vibrissæ, and of the auditory meatus, tragi, are placed vertically in the skin; with these exceptions, the follicles lie oblique to the surface. This obliquity is most marked where the hairs are longest.

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[1] H. Lederman, *Berl. Klin. Wochensch.*, 1903, page 332. "A girl, twenty-two years old, shed her hair every winter. In summer it would grow again. Last winter she became entirely bald and this summer her hair did not grow as usual in spite of treatment. Severe leucoderma is present on the body; this began as circular patches when she was twelve years old."

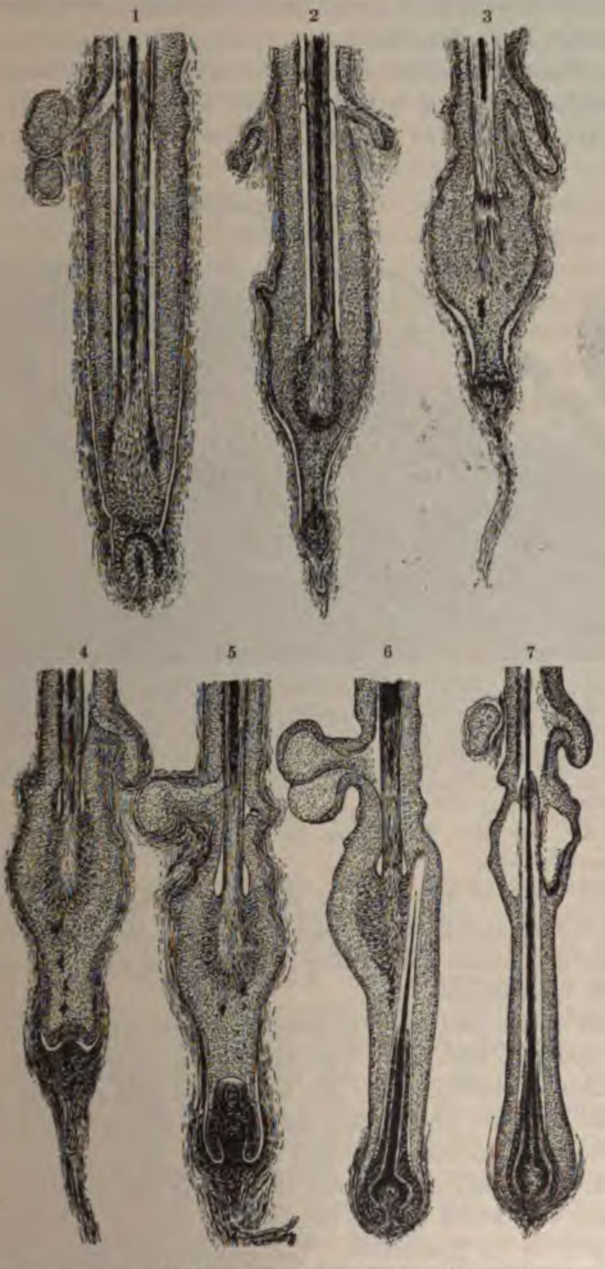

FIG. 22.—REGENERATION OF A HAIR. Only the follicles of the inclosed portion of the hair shafts are represented. The various stages are numbered in order. (Unna.)

The hair follicles follow in arrangement the lines of cleavage of the skin. In certain locations the arrangement is in whorls, beginning at the center and spreading in spiral lines therefrom. The most familiar of these whorls is on the vertex.

Nails.—The nail consists of the nail proper and of a bed upon which it rests and from which it is formed. The nail proper is called the nail plate and consists of: (1) the root, the posterior part that lies under the skin and terminates in a slightly concave posterior border; and (2) the body, the part which lies in front of the root and terminates in a convex free edge.

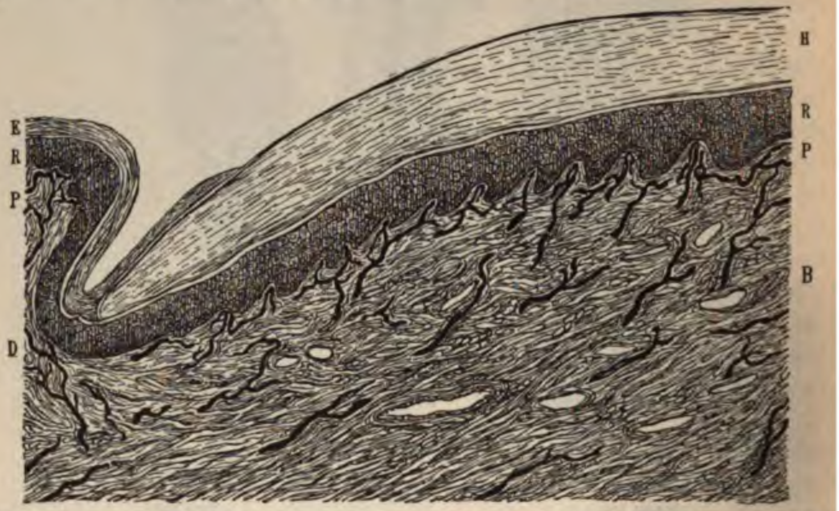

FIG. 23.—NAIL; LONGITUDINAL SECTION. Magnified 100 diameters. *H*, Horny layer of nail; *R*, *R*, rete mucosum; *P*, *P*, papillary layer; *B*, bed of nail, blending with periosteum; *E*, epidermis; *D*, derma with injected blood vessels. (L. Heitzmann, Morrow's System.)

The nail plate is imbedded in the skin on three sides, which are called the lateral and posterior nail walls. On the posterior wall the horny epidermis passes for a short distance out upon the nail plate, forming the eponychium.

The root of the nail plate is inserted posteriorly into the nail fold and rests upon the nail bed. The nail fold is a cleft in the skin, the upper surface of which is called the roof of the fold and the under surface the nail matrix. The surface upon which the nail body rests anteriorly to the matrix is called the nail bed (Fig. 24).

MATRIX.—The nail grows from the matrix; its growth is forward over the nail bed, which simply furnishes it support and does not take an active part in its production. The compression which is necessary to the flattening and linear arrangement of the cells of the nail is furnished by the nail fold, which thus performs a function similar to that of the external root sheath of the hair follicle. The nail matrix extends forward under the posterior fifth of the nail and is directed forward and slightly downward. It projects somewhat beyond the posterior wall, showing through the nail as the lighter colored crescentic area which is called the lunula. The matrix consists of a prickle-cell layer composed of relatively large cells, and at its under surface the papillary structure is well marked. The matrix proliferates from a basal layer of cylindrical cells; above these there are three to five layers of polyhedral cells, and above these several layers of flattened cells, with shriveled nuclei and fine granules in the protoplasm. These are succeeded by the true nail cells. The granules in these cells are finer than ordinary keratohyalin granules and their nature is somewhat uncertain. At least, like keratohyalin granules, they are a part of the cornification process which ultimately produces the horn substance of the true nail cells.

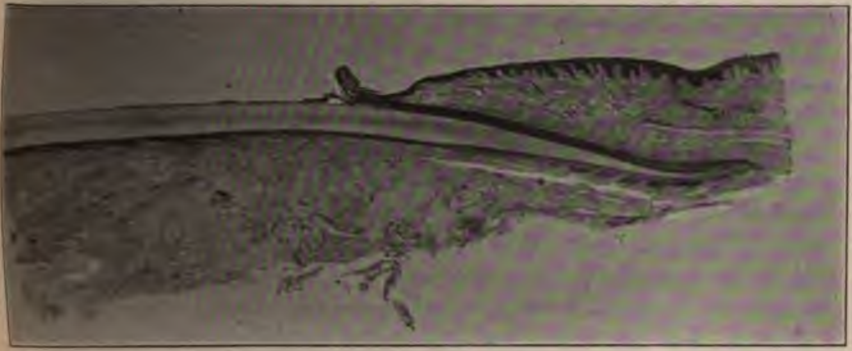

FIG. 24.—LONGITUDINAL SECTION OF NAIL. × 18. (Harris' preparation.)

NAIL BED.—The nail bed is the mucous layer upon which the nail rests in front of the matrix. It has a basal layer of cylindrical cells, and above this two or three rows of polygonal cells, which become flattened, but do not become cornified. Its uppermost cells are in close contact, but are not in organic connection with the nail plate. In front the nail plate does not reach quite to the free border of the nail, but is limited by the horny epidermis of the tip of the finger, which passes up under the nail plate and joins it, forming the hyponychium.

NAIL PLATE.—The nail plate is made up of flat cornified cells. Near the matrix they are flattened, faceted cells arranged in lamellæ. This lamellar arrangement persists throughout the nail plate, but the structure becomes so dense near the free border that it forms an almost homogeneous plate. In longitudinal sections the under surface of the nail plate is plain, but on transverse section it is seen to be ridged from the presence of fine, parallel, longitudinal ridges which fit into corresponding depressions in the nail bed. The process of cornification in the nails differs from that of the horny epidermis generally in the absence of keratohyalin and eleidin from the cells of the matrix, in the production of a harder horn substance than that of the horny epidermis, and in that these nail cells retain their nuclei or nuclear *débris.*

The corium which surrounds the various parts of the nail bed varies strikingly in its papillary structure. That of the roof of the nail fold is without papillæ. Beneath the matrix the papillæ are well marked and large. Beneath the bed of the nail there are no papillæ, but their place is represented by fine ridges parallel to the axis of the finger. On the tip of the finger, where the nail bed ceases, the very marked papillary structure of the finger tip begins. The collagenous-tissue framework of the corium underlying the nail bed and matrix has two distinct arrangements which are peculiar to these parts. One set of bundles radiates vertically from the periosteum to the under surface of the mucous layer, and thus binds the nail very firmly. Another set of fibers is arranged in a horizontal plexus. The elastic fibers are less abundant than ordinary under the nail bed, but their number is increased in the nail matrix.

The nails, unlike the hair, are not normally shed. Krause estimates the growth on the fingers as .08 mm. and on the toes .04 mm. per diem. Observations of mine on the finger nails have closely confirmed his estimate. Their growth is more rapid in summer than in winter and is said to be more rapid in children than in adults. Zeisler has confirmed the interesting tradition that the nails grow less rapidly than normal on a limb which is fractured, but attributes it to the interference with the circulation produced by the bandage.

II.—PHYSIOLOGY [1]

Nutrition of the Skin.—The nutrition of the skin, like that of all other tissues, is supplied by the arterial blood. The interchange of nutritive material and of waste products between the blood and the cutaneous tissues takes place through the walls of the capillaries. As we have seen, these capillaries do not have a muscular coat, so that they are not subjected to vasomotor regulation, and the condition of their blood supply is determined by that of the deeper blood vessels which are under vasomotor control. The vasomotor control of the cutaneous blood vessels consists of a vasoconstrictor mechanism and a vasodilator mechanism, the rôle of which is supplementary to that of the vasoconstrictor mechanism. Both of these are under central nervous control, but are liable to peripheral aberrations. The chief vasoconstrictor center is located in the medulla; in addition there are subordinate centers capable of independent action throughout the cord. The vasoconstrictor fibers pass out in the anterior roots of the spinal nerves, but turn aside to join the sympathetic chain, and, after a longer or shorter course through the sympathetic, join the peripheral nerve trunks and pass to the subcutaneous tissue. The vasodilator centers are said to be evenly distributed through the cord and medulla, and the vasodilator fibers are supposed to pass directly to their destination with the motor fibers. The tone of the cutaneous blood vessels is normally dominated by the vasomotor centers, but in addition to the vasomotor centers there are vasomotor mechanisms at the periphery which, on occasions, may act quite independently of the dominating centers. Such independent action is seen when the vascular tone is disturbed violently by local irritation, as from an insect bite.

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[1] Darier, "La Pratique Dermatologique."—v. Ziemssen, "Cyclopedia of Practice of Medicine."—Duhring.—Foster, "Text Book of Physiology."

The vasomotor mechanism is highly sensitive and is affected by various influences: by direct action on the centers or on the peripheral mechanism of substances in the blood; by reflex or peripheral stimulation, as the application of cold to the surface; by various forms of cerebral stimulations, as the emotions. The mode of action of these influences is exceedingly complex, and it is often impossible in pathological conditions to decide whether the vasomotor disturbance is of the central mechanism or of the peripheral. These disturbances of the vasomotor apparatus of the skin are of the highest pathological importance.

In addition to the control of the nervous system over the nutrition of the skin by regulating its blood supply, the nervous system exercises a direct control upon the nutrition of the skin, as upon that of all other organs. As to the so-called "trophic" nerves, little is known directly. Our chief knowledge comes from the evidence produced by pathological conditions. Such evidence of the direct influence of the nervous system upon the nutrition of the skin is constantly brought to one's attention in the study of cutaneous diseases. The occurrence of bullæ in the distribution of a nerve which is irritated, the acute bedsores of cerebral apoplexy and of degenerative diseases of the cord, the bullous lesions occurring in cerebral degenerative diseases; these are but a few among the many striking evidences that are constantly forcing upon us the conviction of the direct influence of nervous control upon the nutrition and health of the skin. Analogous lesions occurring in other structures, such as the Charcot's joints of locomotor ataxia, confirm this conviction. It is desirable to emphasize the importance of the control of the nervous system over the nutrition and health of the skin, because of the tendency which exists in these objective days to minimize or overlook such factors.

Although there can be no doubt of the importance of the trophic function of the cutaneous nerves, we have practically no knowledge of distinct trophic nerves as such, nor of strictly trophic centers. As far as we know, the trophic function is not a specialized function of any distinct set of nerves or of any distinct nerve center.

As has already been said, the epidermis receives its nutrition from the lymph which comes up from the corium and fills the interepithelial spaces, so that its nutrition is directly dependent upon the blood supply of the corium.

A word needs to be said of the nutrition of the nails and of the hairs. The nail gains all of its nourishment through the matrix from which it grows. The nail plate, after it is completely cornified, is a horny structure without vitality, and receives practically no nourishment from the nail bed. The same statements are almost certainly true of the hairs. The hair is nourished from the papilla from which it grows, and receives nothing from the layers of the follicle. All that has been said of the influence of the nervous system upon the nutrition of the skin applies to the epidermis and its appendages.

Functions of the Skin.—The skin is: (1) the protective covering of the body; (2) an organ of secretion; (3) to a very slight extent an absorptive membrane; (4) the seat of the sense of touch; and (5) it performs very important functions in the regulation of the body temperature.

Protective Function.—The function of the skin as a protective covering has been considered while studying its structure and needs only to be briefly referred to here. Without discussing the useless question of which is the most important function of the skin, it may at least be said that all of the functions and qualities of the skin center themselves around its function as a protective covering. The mechanical pad to act as a buffer against external violence is furnished by the subcutaneous tissue. The horny epidermis furnishes the insensitive structure capable of bearing contact with the external world. The highly resistant chemical and physical properties of the keratin of which it is composed have already been considered. The horny layer is not only capable of resisting all except extraordinary physical attacks, but is unaffected except by the most active chemicals, and is able to prevent the invasion of bacteria. The resistant characteristics of the horny layer are greatly increased by the secretions of the skin which are poured over it. The presence of fat is especially important in increasing its imperviousness, both to the passage outward of the body fluids and to the absorption of water and other substances brought in external contact with it. The weakest points in the skin, considered as a protective coat, are the pilosebaceous openings, and it is through these that most substances, including bacteria, which penetrate the skin, gain entrance. These points, however, are rendered less vulnerable by the masses of sebum which ordinarily occlude them. It is interesting to observe in this connection that the epidermis not only furnishes the protective covering of the body, but that the defensive weapons of animals—horns, claws, teeth, shells, and spurs—are all either of epidermal, or at least of epiblastic origin.

Absorption by the Skin.—The protective functions of the skin are in direct opposition to any qualities it possesses as an organ of absorption, and, as a matter of fact, its absorptive qualities are negative; that is, it is not a perfect protective coat, and hence does not absolutely prevent the entrance of any trace of every substance that comes in contact with it.

The healthy skin is practically impervious to water and to substances dissolved in it; for example, the body may be immersed in a continuous bath without any effect upon the thirst or any diminution in the amount of water drunk and without evidence of the absorption of substances dissolved in the bath. Alcohol and alcoholic solutions are likewise not absorbed by the unbroken epidermis. Fats are absorbed to a certain extent through the epidermis. Where the fat is simply brought in contact with the skin without friction, its absorption is very slight, but, according to Aubert, can be demonstrated after two and a half to five hours. Under these conditions liquid fats are more readily absorbed than solid. When friction is used the absorption of fat is much more rapid and can be demonstrated after five to ten minutes' vigorous rubbing. Under friction solid fats are absorbed more readily than liquid. As is well known, medicines mixed with fats may be made to penetrate the skin. The most familiar application of this fact is the inunction of mercurial ointment. In the absorption of fats, and the same possibly holds true of other substances absorbed by the skin, penetration takes place chiefly through the pilosebaceous openings. Solids, as such, are probably not absorbed by the skin. If they are absorbed it is only after they have been liquefied. Gases are undoubtedly able to pass through the epidermis to a slight extent, but doubtless vary in this capacity. Volatile substances, such as turpentine, ether, chloroform, the essential oils, guaiacol, iodine, are absorbed to a greater or less degree; such of these as are soluble in both water and fats are more readily absorbed.

Cutaneous "Respiration."—Cutaneous respiration, such as occurs in frogs and other amphibians, or of a character to be compared with that of the lungs, does not exist in man. A minute quantity of oxygen is absorbed by the skin, and a similarly small quantity of carbon dioxide is given up. Estimates of the amount vary, but roughly they may be put for both oxygen and carbon dioxide at about $\frac{1}{180}$ to $\frac{1}{200}$ part of that passing through the lungs.[1] The excretion of carbon dioxide is increased by exercise and high temperature. Probably most of the carbon dioxide given off by the skin is in solution in the sweat. There is no evidence of absorption of carbon dioxide in persons immersed in water charged with it.

Williams (*Trans. of the American Association of Pathologists and Bacteriologists,* 1909) reports upon the examination of the sweat glands in seventy cases of nephritis:

"Various conditions were found, including desquamation of epithelium, cystic dilatation of tubules, moderate dilatation of ducts, atrophy of tubules, and the presence in tubules of material resembling casts as found in the kidney. Thrombosis of arteries and veins and arteriosclerosis of the arteries of the skin were also present, the latter condition in fourteen cases. A condition that may be of importance was the apparent formation of two or three layers of cells in the tubules, or at least an increased number of cells if not in regular layers. The actual condition was difficult to determine, but the impression obtained is that the process is an actual multiplication of cells, which does not, however, prove that there is a functional hypertrophy of the glands."

Cutaneous Secretion.—The secretory activity of the skin is comprised chiefly in the secretion of sweat and sebum; in addition to this there is the constant, slight desquamation of horn cells from the surface of the epidermis. The desquamation of epidermis can hardly be regarded as a secretion, but is rather the mechanical detrition of dead matter from the constant friction with external objects. The daily loss of epidermis from the surface of the skin is normally almost inappreciable. In certain diseases it becomes very great, amounting to a gallon or more of scales in twenty-four hours.

SWEAT.—The excretion of sweat is constant. Under ordinary conditions, however, it evaporates as rapidly as formed, and is spoken of as *insensible perspiration*; under conditions of active production it accumulates upon the surface as *sensible perspiration.* The total quantity in twenty-four hours of course varies greatly. According to Darier the total quantity in twenty-four hours is about 1,300 c.c., or a little less than the normal quantity of urine, but under conditions of violent exercise it may amount to as much as 400 c.c. per hour. According to Seguin, the total quantity is about 600 c.c. in twenty-four hours. There is a sort of physiological balance between the amount of water excreted by the sweat glands and that excreted by the kidneys, and a similar balance exists in pathological conditions between the sweat apparatus and the intestines.

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[1] Gerlach estimates the quantity of oxygen at $\frac{1}{137}$ part, of carbon dioxide at $\frac{1}{28}$ to $\frac{1}{92}$; Regnault, of oxygen at $\frac{1}{180}$, of carbon dioxide at $\frac{1}{220}$ part of that of the lungs. Rheinhard estimates the quantity of carbon dioxide at 2.23 grms. per diem, Aubert at 3.87 grms. per diem.

Sweat, when uncontaminated, is of slightly alkaline reaction, but, as ordinarily obtained, mixed with fatty acids of the sebum and epidermis, it shows a slightly acid reaction. Its specific gravity is low—about 1.004. Its chemical composition is approximately as follows:

Water	990.0
Urea	.5 to 1.0
Other extractives	4.5 to 4.0
Sodium chloride	4.0
Other mineral salts	1.0
	1,000.

It contains also a minute quantity of fat and traces of various fatty acids—lactic, butyric, caproic, caprillic, acetic, formic, valerianic. It contains no uric acid or urates.

In pathological conditions the excretion of urea may be greatly increased, and in diabetes excretion of sugar by the sweat may occur. In pathological conditions sweat may contain reddish or bluish pigment derived from the blood or from bacteria. Certain substances when ingested are partly eliminated through the sweat. Among these are arsenic, mercury, sulphur, iodine, bromine, alcohol, ipecac, opium, the balsams, and essential oils. The elimination of bacteria through the sweat in septic conditions has been suggested but not demonstrated, and its occurrence is doubtful.

Sweat is ninety-nine per cent water, and it is evident that its rôle in elimination is relatively small, if the excretion of water is left out of consideration. Even where the excretion of urea or of sugar in pathological conditions reaches the maximum, the total amount only equals that contained in a small quantity of normal urine. The elimination of toxins by the skin seems to be equally unimportant, and experiments in causing retention of sweat and upon its toxicity do not add to the evidence of its importance as an emunctory. As is well known, when the entire surface of an animal is varnished, certain symptoms manifest themselves, as dyspnœa, cardiac distress, trembling, convulsions, subnormal temperature, and possibly death; but these symptoms are not the same for different animals, and they are much more pronounced in small animals, like rabbits and guinea pigs, than in hogs, dogs, and monkeys. And a consideration of the experiments leads to the conclusion that the important factor in the production of the symptoms is the interference with the heat regulation of the body rather than the retention of toxic products. The experiments upon man in this field do not tend to show the danger of retention of sweat. The extensive application of occlusive dressings in pathological conditions of the skin are the cause of no inconvenience in this direction, and in universal dermatoses it is very rare to find systemic disturbances which can be imputed to the failure of the excretory function of the skin.

There can be little doubt that the chief purpose of the production of sweat is the elimination of water. This elimination of water is of great importance in two directions: first, for the regulation of the temperature; second, in softening the horny epidermis and maintaining its pliability. In its passage through the epidermis the sweat percolates through the intercellular spaces, and on the surface of the epidermis it spreads out in an imperceptible film. The importance of this function of the sweat in lubricating, as it were, the epidermis, is constantly manifested in the conditions where the secretion of sweat is greatly diminished or in abeyance, as in ichthyosis. In this connection it must be said that Unna regards the sweat glands as the chief or sole source of the production of fat in the epidermis, but the view is not widely accepted and seems incredible when one considers the amount of fat produced by the sebaceous glands.

The secretion of sweat is a true secretion, analogous to that of the renal secretion, or, more closely, to that of the salivary secretion. It is an entirely different secretion from that of the sebum. In the formation of sebum the cells degenerate and are destroyed to form their product, while in the sweat glands the sweat is the product of active, persisting cells.

The secretion of the sweat takes place in the coil of the sweat gland and is under control of the nervous system. This secretion is controlled by two sets of nerve fibers: first, special sweat excitor fibers which control the activity of the coils, and, second, motor fibers which supply the muscle cells around the tubules, and which, when stimulated, cause expulsion of the sweat. The secretion of sweat is controlled by a center in the medulla which is bilateral and may be unequally irritable, as shown in the rare cases of unilateral sweating. In addition there are subordinate centers in the spinal cord. The sweat nerves follow the rami communicantes to the sympathetic, and then pass into the mixed nerves of the extremities (v. Ziemssen). The secretion of sweat is not only controlled by central influences, but there is a peripheral mechanism which may excite the secretion of sweat independently of central influences. Pilocarpine, for example, will excite sweating at the periphery after section of the nerve trunk supplying the part.

The secretion of sweat is susceptible to various influences: peripheral or central impulses acting upon the nervous mechanism, or substances in the blood which act directly upon the sweat apparatus. A common and manifestly a purposeful excitor of sweat secretion is external heat. The stimulation of sweat secretion by high surrounding temperature is apparently not due to the increased temperature of the blood, but is a stimulation produced reflexly by peripheral sensations. Increased temperature of the blood does not seem to be a particularly strong factor in stimulating the production of sweat, as shown by the absence of increased sweating in many febrile conditions. The sweating produced by exercise is equally as common and as purposeful a manifestation of the stimulation of sweat secretion. Here, likewise, the stimulation does not seem to be the result of increased blood temperature, for the temperature of the blood is not appreciably raised—the prevention of that is the very purpose of the increased sweating. In this case the stimulation is probably the result of sensory impulses arising from the muscular exercise, or perhaps from the presence in the blood of waste products of muscular activity which act upon the sweat centers. Less commonly, but equally positively, the sweat centers are affected by impulses of cerebral origin. This is seen in the influence of the emotions, as embarrassment, in stimulating sweating. Violent nervous shock, either central or peripheral, may act in the same way, and the stimulation thus produced may be so strong as to cause the abundant production of sweat quite independently of thermal conditions. Such cold sweating is a common manifestation of severe nervous shock or physical depression. The normal tone of the sweat apparatus may be disturbed in various conditions of moderate physical or nervous depression, which result in persistent increased sweating of various parts.

The stimulation of the sweat apparatus by substances in the blood acting upon it is seen in the exhibition of numerous drugs and probably in various pathological conditions. The toxins of various pathogenic bacteria probably act in this way, as shown, for example, in the increased sweating in articular rheumatism and in septicæmia. Many drugs act in the same way; pilocarpine is the most characteristic example of these, but opium, acetanilide, antipyrine, and various coal-tar preparations illustrate the same phenomenon. Other drugs, as atropine, *per contra*, inhibit the secretion of sweat.

Sebaceous Secretion.—The sebum or sebaceous secretion is fluid or semifluid in the glands, but tends to harden in the duct, where it may become of cheesy consistence. Frequently it accumulates in the gland and duct and may be expressed in a wormlike mass. It consists of the fatty substance of the cells mixed with cellular *débris* and sometimes pigment. Often there is found in the gland an acarus, the demodex folliculorum, which does not ordinarily produce any pathological change. According to Darier, the sebum consists of two thirds water and one third fatty substance, which is made up of fats (palmatin and olein), fatty acids, soaps, cholesterin, an albuminoid similar to casein, and alkaline chlorides and phosphates. The sebum furnishes an abundant supply of fat which lubricates the hairs and adds to their suppleness and impermeability.

Nerve fibers have not been traced to the cells of the sebaceous glands, and it is generally held that the sebaceous secretion is not under the control of any nervous mechanism. Duhring, however, believes that it is, and that its activity is influenced in about the same way as that of the sweat glands. The sebaceous secretion is influenced by temperature and blood supply; it is greatly increased during the period of adolescence, when the growth of the hair is most active; it is also increased in certain pathological conditions; but the sebaceous glands do not show the violent fluctuations in activity under temporary stimuli that the sweat glands do, and the evidence in favor of nervous control over their secretion is not strong.

The Sense of Touch.—The sense of touch is a special sense; its sensations are distinct from the sensations of general feeling, which arise from the stimulation of the sensory nerves, and require for their production special terminal organs. The terminal organs of the sense of touch are located chiefly in the skin, but they occur also in the mucous membranes lining the various passages of the body for a short distance from the orifices.

The sense of touch includes two classes of sensations: first, those arising from mechanical stimulation; and second, those arising from thermal stimulation. The first of these classes is sometimes divided into (1) perception of pressure and (2) touch proper, which appreciates particular qualities, as roughness or smoothness, softness or hardness, dryness or dampness; but it is exceedingly doubtful that all of these perceptions are not contact perceptions depending wholly upon variations in pressure. It is impossible to distinguish between what is called mere contact and touch in which the element of pressure comes in.

Pressure or touch perception, and thermal perception are independent of each other; the acuteness of the two does not correspond in all parts of the body, and one of them may be disturbed without corresponding disturbance of the other in the same area. Thus temperature sensation may be impaired or absent in an area without any impairment of touch perception, or the contrary may occur. These and other facts strongly suggest that there are two distinct kinds of terminal organs in the skin, one for touch perception, the other for temperature perception, and it is probable that they are connected with different parts of the central sensory organs by separate nerve fibers which pursue different courses in the spinal cord. The two perceptions, however, are closely associated and frequently overlap and confuse each other. Thus a cold or a hot substance placed upon the skin where the touch sensation is not very acute will appear heavier than the same substance at the body temperature. As to which are the terminal organs of the thermal sense and which are the organs of the touch sense, we are not in a position to make definite statements. The classical assumption is that the so-called touch corpuscle of Meissner and the Pacinian bodies in the subcutaneous tissue are the terminal organs of pressure or touch perception, and that the free nerve endings are the terminal organs of temperature perception. The rapidity of the perception of heat and cold suggests that the terminal organs for these sensations are very near the surface—that is, in the epidermis—while the organs of pressure sensation might lie deeper. And this suggestion points toward the free nerve endings in the epidermis as terminal organs of temperature perception. But while some of these may be organs of temperature perception, others are probably organs of touch perception.

The sense of touch is affected by no external stimuli except those produced by pressure and temperature. Like other special senses, however, its specific sensation may be excited under abnormal conditions by cerebral impulses, as is seen in the occurrence of sensations of heat or formication due to central stimuli.

The sensations of *tickling* and *itching* are confined to those parts endowed with the sense of touch, and there would seem to be no reason to doubt that they are aberrations of that sense. Although it is held that we are without satisfactory explanation for their occurrence, a consideration of their character and their causation seems to indicate clearly that they are due to exaggerated stimulation of the terminal organs of the sense of touch, and that they are thus analogous to pain in the sensory nerves generally; that, as pain is an exaggeration of the sensation of general feeling due to overstimulation of any sensory nerves, so tickling, and more especially itching, are exaggerations of the touch sensation due to overstimulation of the terminal organs of the sense of touch.

The temperature sense is most acute on the face, the lips, the cheeks, and the temples, and, according to Nothnagel, on the forearms and arms, where a variation of .2° C. is appreciated. The least sensitive parts are the legs and the front and back of the trunk. Where it is least acute a variation of less than 1.2° C. is inappreciable. The temperature sense is most acute for objects between 27° and 33° C. With extremes of heat and cold thermanæsthesia is produced. It is an interesting fact that the acuteness of the perception of heat may differ from that of the perception of cold in the same area. Thus the glans penis and the clitoris are almost or quite insensitive to cold, but not to heat. The temperature sense appreciates only variations from the temperature of the surface of the skin. Therefore its judgment of the temperature of bodies is only relative. When the hands, for example, are cold, cold substances appear less cold than usual, and hot substances hotter. In the same way substances that are good conductors of heat seem hotter or colder than substances of the same temperature, which are poor conductors of heat and therefore part with or absorb heat less rapidly.

The variations in the acuteness of the perception of touch proper are wider than those of temperature perception. The variations of different parts of the body in this respect are shown in the following table taken from Weber, in which the figures represent the minimum distance for the perception of two points of a compass applied to the skin:

Tip of tongue	1.1 mm.
Palm of last phalanx of finger	2.2 "
Palm of second phalanx of finger	4.4 "
Tip of nose	6.6 "
White part of lips	8.8 "
Back of second phalanx of finger	11.1 "
Skin over malar bone	15.4 "
Back of hand	29.8 "
Forearm	39.6 "
Sternum	44.0 "
Back	66.0 "

Both the temperature sense and the touch sense proper are capable of development. The temperature sense, perhaps, cannot be developed by experience, but the judgments derived from that sense are capable of distinct development; thus bath attendants with practice become able to determine very accurately the temperature of water by touch alone. Touch sense proper is capable of very great development by practice. This is seen particularly in the blind, who are compelled to depend so much upon the sense of touch, and in individuals whose occupations compel them to rely largely upon the sense of touch in forming their judgments. Thus hair dealers determine the quality of hair chiefly by the touch (Duhring), and Erasmus Wilson relates the case of a buyer of silk who was able by touch alone to distinguish forty varieties of that article. The improvement by exercise of the sense of touch is explained not by an increased development of the terminal organs, but by a more exact limitation of the sensational areas in the brain (Foster).

Sense of Locality.—The power of correctly localizing the sensations of touch, which may be called the sense of locality, is derived from experience. The young infant will not definitely locate a touch perception, as it will not locate a pain; it is only by the accumulation of experience that judgment develops as to the location of these sensations. Occasionally abnormal conditions of the sense of locality are met with. Thus

Stirling reports a case in which cold water applied to the scalp produced a sensation referred to the skin of the loins, and Landois a case in which pricking of the skin over the upper part of the sternum produced a sensation in the knee. It is not infrequent for a sensation on one side to be referred to a corresponding part on the other. We see this confusion of locality frequently in pathological conditions where an abnormal sensation, particularly itching, arising from a lesion at one point is referred not only to that point, but to the corresponding point upon the other side. Where the sensation is very acute at one point a corresponding sensation may be referred to widely distributed parts.

Heat-Regulating Function of the Skin.—For the maintenance of a fixed temperature, such as man and other warm-blooded animals have, there must be an apparatus for the dissipation of heat of wide range of activity under varying thermal and metabolic conditions. This apparatus is furnished by the skin and the lungs. In animals that are covered with a thick coating of fur or hair, which renders the rapid dissipation of heat through the skin impossible, the chief channel of its loss is the lungs. In man, of the heat lost by the body, about seventy-seven to eighty-five per cent is lost through the skin, about nine to twenty per cent through the lungs, and from three to six per cent through the fæces and the urine. The skin is thus the great organ in man for the dissipation of heat and the regulation of temperature.

The loss of heat from the skin occurs by radiation and conduction and by evaporation of sweat. The two means of regulating this loss are: first, the variable blood supply of the skin, and second the variable secretion of sweat, both of which are, as we have seen, under nervous control. These two, although independent of each other, ordinarily act in unison. When there is active production of heat by exercise or other metabolic processes, or when the body is surrounded by a warm medium, the sweat-secreting apparatus becomes active and the vasomotor mechanism dilates the blood vessels of the skin. By the first of these processes there is an outpouring of sweat upon the surface, through the evaporation of which much heat is consumed; and by the second an increased quantity of heat is carried in the blood to the skin, where its dissipation becomes possible through radiation and conduction. Under opposite conditions, the secretion of sweat stops and the blood vessels of the skin contract, and thus the loss of heat from the surface is reduced to a minimum. In the emptying of the cutaneous blood vessels the vasomotor mechanism is aided by the contraction of the involuntary muscles of the skin, which by their contraction under cold increase the tension of the skin and the pressure around the blood vessels. This is manifested in the familiar phenomenon of goose flesh (*cutis anserina*) under exposure of the skin to cold.

The most elastic of these means of dissipating heat is the secretion of sweat. When this is abundant and its evaporation rapid, it furnishes a means for the dissipation of heat equal to almost any emergency; thus one can stay in a dry atmosphere in a chamber heated to over 250° F. almost without discomfort and without any perceptible change in the body temperature, so long as the secretion of sweat is active.

III.—ETIOLOGY [1]

It is evident from a consideration of the physiology of the skin that its health may be affected:

(1) By causes which act directly upon the cells of the skin.

(2) By causes which affect either the supply or the composition of its nutrient fluids, the blood and the lymph.

(3) By causes which influence the nervous apparatus which is concerned in the nutrition of the skin.

The causes which are included in group (1) are for the most part external, that is, they produce disturbances in the skin by their direct effect upon its cells; but even here the results may not be limited to the area directly subjected to the exciting agent, for, through the intervention of the nervous system and perhaps at times of the circulatory system, lesions may be excited at distant points as a result of the primary lesion. The causes affecting the skin through its blood and lymph supply and through its nervous control are usually internal. They are almost innumerable, and they are often very obscure.

It is, of course, true that in many, if not in most instances, the causative factors of a dermatosis are not simple in their action and cannot be sharply classified in one of these groups. For example, an irritation of the skin from the ingestion of an iodine salt is probably due to the direct action of the chemical upon the cells of the skin, but it reaches these cells through the blood; and so here we have a pathological process which is due to the direct effect of an agent upon the tissue cells of the skin, but which reaches these cells through its absorption first into the blood. In the same way the wheals and the itching which sometimes follow the ingestion of quinine or morphine are probably due to the effect which the drug has upon the peripheral vasomotor mechanism of the skin; so that here we have lesions which are produced by alteration in the blood and at the same time by disturbances of the nervous control of the skin.

From another standpoint the causes of diseases of the skin may be classified according to their intensity as predisposing and exciting. A predisposing cause may be defined as one which, while not intense enough to excite an actual pathological process, lowers the normal resistance of the tissues and renders them more vulnerable to pathogenic agents. Predisposing causes may thus differ from exciting causes only in degree. External causes may be predisposing as well as internal. A superficial abrasion which permits of infection with the specific poison of syphilis or of actinomycosis is as much a predisposing cause of disease as the lowered condition of health which allows the tubercle bacillus to obtain a footing. Usually, however, in speaking of predisposing causes we refer to those conditions of the general health which make the individual less able than normally to resist pathological processes. The predisposing causes of this sort are numerous.

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[1] Duhring.—MacLeod.—Darier. "La Pratique Dermatologique."

Individual Susceptibility, Idiosyncrasy, Predisposition, Diathesis.—The natural tolerance of the skin varies considerably in different individuals. The greater tolerance of one individual as compared with another may frequently be explained by differences in the texture of the skin; the fine, thin, dry skin is naturally less able to resist external irritants than the thick, greasy skin. The reasons for variations in vulnerability are frequently, however, not apparent. Certain individuals, for example, are almost invulnerable to the attacks of most cutaneous parasites, while others show an abnormal irritability to them. Again, in certain individuals the irritability of the skin to certain agents amounts to marked idiosyncrasy. Thus Stelwagon has recorded the case of a man who was so susceptible to quinine that the application of a minute quantity in the form of a hair tonic caused a violent eruption. Idiosyncrasies to various drugs are by no means rare.

Diathesis is a term with which there has been much conjuring, and, like many vague terms, it has served its purpose as a cloak for inaccurate impressions. Hutchinson, in considering what he called the "pedigree of diseases," has defined diathesis as "any condition of prolonged peculiarity of health giving proclivity to definite forms of disease." In this sense diathesis is synonymous with predisposition or susceptibility, whether inherited or acquired. And of the existence of this we have constant evidence. Such a factor is indefinite and uncertain, because it is impossible to agree upon what is the standard of resistance to disease of the normal individual; but there is no room for doubt that certain individuals fall well below the average in their capacity for resisting disease. They have unstable nervous systems, they are the prey of drug habits, of tuberculosis, and of other bacterial diseases. Such diathesis or predisposition is usually the result of poor ancestry, as shown by too intimate marriages among ancestors, or by a neuropathic family history, or by a family history of tuberculosis or carcinoma, or by some other evidence of tissue weakness. An individual predisposition or lowered resistance of the same character which practically amounts to diathesis may be the result of unfavorable circumstances, as a childhood of hardships or of forced development, or a too hard struggle for existence, or an existence under unsanitary conditions. Individuals presenting such diathesis or predisposition show an undue susceptibility to dermatoses as they do to other pathological processes.

There are certain peculiarities of the skin in individuals and families which, quite apart from any condition of the general health, tend in the direction of certain dermatoses. Just as the complexion and the color of the hair and eyes are apt to be transmitted to descendants, so certain peculiarities of the skin may be transmitted. For example, certain individuals inherit the type of skin which has a very abundant development of sebaceous glands, and they are almost sure during adolescence to be affected with severe acne and comedones. Again, the individual who inherits dark skin and hair may inherit a predisposition to pigmentary affections. In the same way we find in the family history of patients an inherited predisposition to psoriasis, to eczema, to keratoses, to epithelioma, to xanthoma, to idiosyncrasies to drugs, or to such rare conditions as xeroderma pigmentosum or multiple benign cystic epithelioma. They do not inherit the pathological processes, but they inherit a family characteristic in their tissues which shows in a tendency to the development of these affections. There exists a similar tendency to the inheritance of congenital defects of the skin, as moles, nævi, icthyosis, epidermolysis bullosa, or congenital defects of the hair and nails. Here the individual does not inherit a skin that is disposed to the development of these defects, but he is born with a skin in which the defect actually exists.

It is in the sense of the inheritance of a predisposition to disease that the rôle of heredity is most important in the etiology of diseases of the skin. Heredity, however, may also be responsible for the transmission of specific diseases. The best example of this is the transmission of syphilis from the parents to the child *in utero*. A similar transmission from the mother to the child may occur in smallpox, scarlet fever, and measles, and perhaps in other infectious diseases; it is uncertain whether it occurs in tuberculosis or leprosy. The list of diseases which may be directly transmitted from mother to child *in utero* is very short.

Race and Nationality.—There are a very few definite racial predispositions. The best established of these is the predisposition of the negro to connective-tissue growths, as shown by the relative frequency of the occurrence of keloids and fibromata and elephantiasis in negroes. According to Balloch this predisposition is also shown to malignant growths, mesoblastic tumors forming a larger proportion of the malignant growths in negroes than in Caucasians. Another tendency which, perhaps, may be called a racial characteristic is that of the Jews to dermatoses of nervous origin. There are many well-recognized national or local tendencies to certain dermatoses. Common illustrations of these are the relative frequency of favus and parasitic diseases in Italy and southeastern Europe; of pellagra in Italy; of leprosy in Scandinavia. There are certain contrasts of this sort which are curious. For example, favus is very common in Scotland; it is quite rare in London; the recently discovered disease blastomycosis has been found in numerous instances in Chicago—is, in fact, not excessively rare—while it is a dermatological curiosity elsewhere. But these national and local tendencies to certain dermatoses have very little to do with predispositions; they are almost invariably the expression of peculiar conditions of life which are favorable to the propagation or development of particular affections.

Age.—Certain diseases show a decided predilection for certain periods of life. Often the reason for this predilection is apparent in some circumstance favorable to the development of the direct causative factors, but frequently the reason is not ascertainable. In infancy the gastro-intestinal tract is very susceptible to disturbances and the nervous balance is exceedingly sensitive; and at that time of life we see frequently the dermatoses which are associated with these conditions, like eczema, urticaria, the erythemas, and simple herpes. The infant skin, also, is very vulnerable to pus-forming bacteria, and so impetigo is common, and septic processes in the skin are accompanied by an undue amount of pus. Certain diseases from their very nature occur in early infancy. Of course the congenital defects, as nævi and icthyosis, usually appear in infancy, as do diseases which are hereditarily transmissible, as congenital syphilis. The exanthemata also usually occur in infancy, because they are so widespread and so contagious that escape from them during the first few years of life is almost impossible. In the cases of chicken pox and German measles there seems to be actually greater susceptibility in childhood, for these two affections do not readily attack adults. The vegetable parasitic diseases are chiefly met with in childhood; ringworm of the scalp and favus are especially, for what reason we do not know, diseases of childhood, and tend to disappear as adult life is reached. On the other hand, tinea versicolor is a disease of adult life. Lupus vulgaris is another disease which usually begins in childhood or adolescence. In this respect it is in contrast with lupus erythematosus, which is apt to develop after twenty-five or at the time when lupus vulgaris tends to get well.

At puberty and during adolescence, when the sebaceous glands together with the hair follicles take on a greatly increased activity, acne and comedones are of almost universal occurrence and seborrhea is common. Early adult life is the usual time for the occurrence of lupus erythematosus, of psoriasis, of rosacea, and of the occupation dermatoses. At the time of the menopause there is a tendency to the development of pigmentary disturbances, to disturbances of the sweat function, and to functional nervous disturbances. Similar nervous disturbances are seen, but less frequently, at that other physiological crisis—puberty.

In old age the skin becomes more or less atrophic, and is accordingly more sensitive to injuries. It also shows a tendency to degenerative processes. An idiopathic pruritus is an exceedingly common affection at this time, keratoses are apt to develop upon the exposed surfaces, and the development of cutaneous carcinomata is frequent. Body pediculosis, perhaps from the increased vulnerability of the skin, is most common at this time.

Sex.—The difference in the relative frequency of various dermatoses in men and women is largely a matter of difference in habits and environment. Men are more apt to have the occupation dermatoses, the dermatoses of venereal origin, epithelioma of the lower lip and mouth as the result of smoking, and, from the same cause, leukoplakia buccalis. There are certain dermatoses associated with pregnancy, like chloasma (the most frequent example) and impetigo herpetiformis. There are a few other diseases, of which lupus erythematosus is the best illustration, which are distinctly more common in women than in men without any evident reason for the fact. In general, according to Duhring, women are more frequently affected with diseases of the generative system, and men with diseases of the urinary system; and women in their susceptibility to diseases resemble children more than they do men.

According to Williams, men are more than twice as subject to carbuncles as women, while neoplasms are more than twice as common in women as in men. This latter statement, however, I am sure does not apply to malignant growths of the skin.

Seasons and Climate.—The effect of seasons and climate upon dermatoses is largely a matter of the influence which they exert upon living surroundings, and of the favorable or unfavorable conditions which they present for the development of parasites and bacteria. Such diseases as are transmitted by direct contact or intimate association of individuals are likely to be more frequent in cold climates and during the winter season. Illustrations of this are seen in the greater frequency of smallpox in winter and of the great increase of scabies, which is seen in public hospitals in large cities during the winter months. Bacterial diseases are most prevalent in hot climates, which furnish the favorable conditions for growth presented by moisture and heat and the accumulation of dirt. Characteristic illustrations of such diseases are the endemic tropical diseases, like mycetoma and Aleppo boil.

Certain diseases are directly traceable to the heat of summer or the cold of winter, while a predisposition to others is traceable with equal directness to the same conditions. The sweat eruptions, like prickly heat, are directly associated with hot weather, while frostbite is directly, of course, the result of cold. The inactive, dry skin of winter is more vulnerable to irritants than the well-lubricated skin of summer, and consequently the development of dermatitis or of pruritus without dermatitis from external irritation is more apt to occur in winter. Chronic eczemas, psoriasis, lupus erythematosus, and other scaly inflammatory dermatoses are apt to be exaggerated during cold weather. Certain dermatoses, again, like herpes zoster and erythema multiforme, are prone to occur during the changeable weather of spring and autumn, because such weather is favorable to the development of the constitutional conditions which underlie these dermatoses.

Occupation and Mode of Life.—These are, of course, influential as predisposing or exciting causes of skin diseases as they involve habits which are harmful to the skin. The influence of unsanitary living surroundings is manifested in their tendency to reduce the vigor of the tissues and to expose the individual to the attack of parasites and microörganisms. The influence of occupation in the production of dermatoses is of considerable practical importance, and is apt to be overlooked frequently unless one is on the alert for it. The dermatoses which result directly or indirectly from occupation are, in general, such as are produced by moderate but long-continued irritation. Thus we frequently see eczemas of the hands in washerwomen, janitors, and bartenders from the constant use of soap and water, in painters from the use of turpentine, in workers in acids and alkalies and dyestuffs—particularly the aniline and arsenical dyes. Manual laborers show callosities and frequently septic infections. Cabmen, sailors, and farm laborers who are much exposed to wind and weather frequently develop a deep erythema of the face, rosacea, and keratoses. Bakers, typesetters, carpenters, salesmen, who stand at their work, develop varicose veins and consequent leg ulcers and eczemas.

It is not unusual also to see dermatoses which are influenced or excited by some avocation or diversion of the patient, and the influence of this is particularly likely to escape us because we are not apt to be on the outlook for it. Perhaps the commonest illustration of such an avocation dermatosis is seen in the eczemas of the hands, which occasionally occur in enthusiastic amateur photographers. A good illustration of this sort I have seen in a lady who for more than a year was treated for what was thought to be an erythematous eczema of the hands, and who discovered quite accidentally, after the fact had escaped several acute dermatologists, that the dermatosis was due to primroses which she cared for.

Exciting Causes.—As suggested above, a predisposing cause of a dermatosis frequently becomes an exciting cause simply by an increase in the intensity of its action, so that most of what has been said under the heading of predisposing causes is to be regarded as supplementary to any further consideration of exciting causes. The exciting causes of dermatoses are naturally divisible into local and constitutional causes, but this sharp division is by no means constantly followed by nature. We see many dermatoses due purely to external causes; we see others due as purely to internal causes; but we also see many in which internal and external causes supplement each other. Indeed, we see few dermatoses of internal origin which are not more or less modified by secondary external influences, as scratching or infection.

Internal Causes.[1]—The internal causes of dermatoses may be divided into (1) toxic, (2) nutritional, (3) nervous. The toxic causes may be further divided into (*a*) drugs and other chemicals, (*b*) foods, (*c*) bacterial toxins, (*d*) infections. The nervous causes may be divided into (*a*) peripheral or central lesions of the nervous system, (*b*) neuroses, (*c*) reflex nervous disturbances, (*d*) emotional disturbances. It is not difficult to present a general classification like the above of the internal causes of cutaneous diseases, and for the purpose of a comprehensive survey of the subject such a classification is useful. It is needless to say, however, that no such accurate differentiation of the causes of dermatoses can be made in practice. In the first place our knowledge is far too limited, and in the second place the causes themselves are too complex.

TOXIC CAUSES.—Toxic substances may affect the skin by entering into the blood and lymph and then coming directly in contact with the cells of the skin, or they may produce their effect indirectly by causing a disturbance of the nervous mechanism controlling the nutrition of the skin. The latter is by far the commoner method of action.

Drugs.—The eruptions caused by drugs are instructive, as illustrating the lesions in the skin which can be produced by toxic substances in the body; for in the drug eruptions we are not dealing with assumed poisons in the blood as the cause of cutaneous lesions, but with poisons which can be definitely traced. The widespread importance of toxic substances in the production of dermatoses is shown by the fact that different drugs cause at times almost all forms of cutaneous lesions. For example, from morphine and valerian we may get intense pruritus without lesion; from belladonna, quinine, the salicylates, chloral, antipyrine, and the antitoxic sera we may get vasomotor disturbances showing as hyperæmic, macular, or papular eruptions or as eruptions of wheals; from the long-continued use of arsenic we may get great thickening of the horny epidermis of certain parts; from iodine and bromine salts we may have the formation of granulomatous tumors. If these various lesions can be produced by drugs, it is not difficult to conceive of analogous eruptions resulting from other toxic substances.

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[1] Bulkley, "Skin Diseases and Internal Disorders," The Rebman Co., 1906.—This classification is adopted with modifications from Darier in "La Pratique Dermatologique."

In the case of certain eruptions produced by iodine and bromine, whose salts are excreted partly through the skin, we have perhaps our best illustration of eruptions produced through direct irritation of the cells of the skin by poisonous substances from the blood. In most of the drug eruptions the cutaneous lesions are evidently the result of disturbance of the nervous mechanism, particularly the vasomotor mechanism controlling the nutrition of the skin. Hence we most frequently see, as the result of the use of drugs, eruptions of hyperæmic macules or patches, of wheals, or of inflammatory papules. But the drug eruptions are by no means limited to these; in fact, in their variety the drug eruptions present at times almost all forms of cutaneous efflorescence.

Foods.—Too little food may, of course, act as a predisposing factor of diseases of the skin by lowering the resistance of the tissues. And we sometimes see such a cause working, as, for example, in sailors or inmates of penal institutions who have not had sufficient food or who have been deprived of the necessary variety. We see a similar situation occasionally in a devotee of some dietetic vagary, as vegetarianism. But the bad effects which are seen as a result of eating are much more frequently due to indulgence in too much food or in indigestible or improper articles of food. Here the effects are, of course, indirect, the vicious habits producing first gastrointestinal, toxic, or metabolic disturbances.

Among the various articles of food that are not infrequently the cause of cutaneous disturbances are strawberries and other small berries, vegetable salads, in which the dressing is usually the peccant factor, pickles, olives, shellfish, sausages, and game which has become "high." A too free indulgence in hot breads, griddle cakes, buckwheat, oatmeal, and other cereals, and in pastries and sweets is frequently a factor in cutaneous disturbances through the bad effect upon the digestion. The same is true of the habitual use of alcoholic liquors with the attendant disturbances of digestion and metabolism which they produce.

Bacterial Toxins.—Examples of eruptions due to the bacterial toxins are seen in septic infections, diphtheria, influenza, cholera, malaria, and in various other infectious diseases. The most familiar eruptions of this sort are those which are an essential part of the symptom complex of typhoid and typhus fever, of secondary syphilis, and of the exanthemata. Eruptions from bacterial toxins can be produced not only b toxins elaborated by bacteria in the patient's body, but also by bacteria toxins introduced from without, as shown by the occurrence of derma toses in connection with the use of the therapeutic sera.

General Infections.—The eruptions of the exanthemata, with th exception of smallpox, are probably illustrations of dermatoses produce by bacterial toxins. This is also probably true of the occasional pr dromal eruption of smallpox. In the typical eruption of smallpox, how ever, the eruption is probably due to the direct attack of the organis of the disease upon the cutaneous cells; the same perhaps holds true the eruptions of secondary syphilis. In the metastatic lesions whic are occasionally seen in the course of septicæmia, we have an illustr tion of the eruptions due to the attack of bacteria which reach the ski from within. This method, however, of production of cutaneous lesio by the direct invasion of the cells of the skin by bacteria from th blood, is certainly not nearly so frequent as that of their productio by indirect action upon the skin through bacterial toxins elaborate elsewhere.[1]

Autotoxins.—We are constantly confronted in dermatology with th problems of metabolic disturbances and of other forms of autointoxic tion, and there can be no doubt of their great importance in the caus tion of cutaneous diseases. But the knowledge at our disposal of th chemistry of these conditions is so meager and so uncertain that we ca seldom do more than consider generalities. We do not know the actu chemical substances which produce the symptoms with which we ar familiar; we can only reason from the broader features of famili clinical pictures. We see situations of this sort in what is called th gouty or rheumatic diathesis, in hepatic insufficiency, in intoxicatio from intestinal disturbances, perhaps in insufficient renal excretion. I these conditions it is impossible from our lack of accurate chemic knowledge to speak definitely of the toxic substances, but of the char acter of the conditions as a whole there is no doubt. There is a definit disturbance of the balance between assimilation and excretion; th assimilating apparatus of the body is not equal to the demands place upon it by overgenerous habits of living, and there results an accumu lation in the body of products not completely elaborated for excretio That such products are the cause of dermatoses we have constant evi dence. These conditions of perverted metabolism, like other toxic dis turbances, produce most frequently vasomotor disturbances in the skin like urticaria, the polymorphous erythemas, and certain forms of eczema

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[1] Riesman, "Malaria and Skin Diseases," *American Med.*, March 22, 1902.—Engman, *Jour. Cutan. Dis.*, November, 1903.

They doubtless also are causal factors in many other dermatoses concerning whose origin we are at present quite in the dark.

There are also certain forms of very definite disturbances of metabolism or of autointoxication in which, as would be expected, we find dermatoses.[1] Thus in diabetes, in pathological conditions of various glands which probably elaborate special secretions, as the thyroid and suprarenals, there are well-recognized accompanying disturbances of the skin. Nephritis also is occasionally accompanied by dermatoses, but the occurrence of this association is less frequent than might be expected in a condition of such profound disturbance of excretion; and this fact suggests that the substances completely elaborated for excretion, which the damaged kidneys fail to eliminate, are not as deleterious as those which have failed of complete elaboration through deficient metabolism. True gout not infrequently has associated with it certain dermatoses, of which eczema is the most frequent. But the association is not common enough to indicate that the uric acid salts are particularly irritating to the skin; and gout, as distinguished from the "gouty diathesis" is not, in the United States, at least, a common cause of dermatoses.

Nutritional Disturbances.—The dermatoses due to nutritional disturbances cannot be separated from those of toxic origin. But there can be no doubt of the importance in the causation of diseases of the skin of the lowered resistance of the tissues which occurs in many nutritional disturbances. All that has been said of the influences of poor ancestry, of unsanitary occupations and surroundings, and of similar circumstances which produce a lowered resistance of the tissues, applies in this connection.

Nervous Causes.—The influence of the nervous system in dermatoses is almost omnipresent. We see it most frequently when, by disturbance of its functions, the nervous system acts as the means through which toxic or other underlying disturbances affect the skin. We have also many illustrations of dermatoses which are due directly to pathological processes in the nervous system. In lesions of the central nervous system, especially of the cord, cutaneous symptoms frequently occur. The most frequent of these is an appreciable change in the texture of the skin. In locomotor ataxia we have the erythemas, urticaria, zoster, bullous eruptions, trophic ulcers, and gangrene; in spinal meningitis, erythematous and vesicular and bullous lesions; in syringomyelia and lepra, anæsthesia and trophic ulcers in addition to the commoner erythemas and urticaria; in lesions of the sympathetic, hyperæmias and anæmias, pigmentary disturbances and increased sweating. In peripheral nervous lesions the manifestations in the skin are common. The most familiar example of such lesions is zoster. In multiple neuritis we have sensory disturbances of the skin with pain, numbness and loss of sensation, and vasomotor and trophic disturbances with erythemas, atrophy, and changes in the skin's texture. We occasionally see vasomotor changes in the skin resulting in permanent vascular and nutritional disturbances, the best illustration of which is found in the local ischæmia of Raynaud's disease. We also see disturbances in the formation of pigment, like leukoderma, and nutritional changes in the hair and nails which, there seems every reason to believe, are of trophic, or in other words, of nervous origin.

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[1] Morrow, "Diabetes and Skin Diseases," *Medical Record*, April 11, 1896.—Sherwell, *Medical News*, June 29, 1901.—Hyde and McEwen, "Exophthalmic Goitre and Skin Diseases," *Amer. Jour. Med. Sci.*, June, 1903.—Waelsch, "Skin Diseases and Metabolic Disturbances," *Monats.*, vol. xlii., No. 10 (abstract of elaborate paper).—Glaserfeld, *Inaug. Dissert.*, Munich, 1904, *Abs. Monats.*, vol. xli., No. 4 (elaborate paper).

In many cutaneous disturbances the influence of nervous reflexes is not infrequently seen in the occurrence of sensory or vasomotor disturbances at points widely distant from the site of original irritation. This is seen in many cases of eczema, herpes, and urticaria, but the most unquestionable illustration of it is seen in those cases of pruritus arising from external irritation at some point in which the itching at the one point is referred to widely distributed parts of the body and perhaps becomes general.

Emotional Disturbances.—The influence of the emotions is seen in many dermatoses. Sensory, vasomotor, and trophic disturbances of the skin may be caused by nervous exhaustion, fright, grief, long-continued anxiety, and similar nervous and mental conditions. To such influences at times can be traced various dermatoses, as urticaria, eczemas, pigmentary changes in the hair and skin, psoriasis, and lichen planus, as well as rarer conditions like dermatitis herpetiformis, pemphigus, and morphœa. The association of various vasomotor and trophic disturbances of the skin with epilepsy and insanity is not infrequent. The only cutaneous manifestations commonly found with hysteria are disturbances of sensation, of which anæsthesia is the classical example. Occasionally, however, there are found bullæ and ulcers, which there seems every reason to believe are of trophic origin; I have recently seen a well-defined zoster of the neck in the case of a man with hyperpyrexia in which competent neurologists were able to make no other diagnosis than hysteria. The most frequent lesions of the skin seen in hysteria, however, are traumatic ulcers and other traumatisms secretly produced by the patients upon themselves.

The foregoing brief survey gives but an intimation of the importance of the nervous system in cutaneous diseases. As we consider the various diseases of the skin we will have constantly impressed upon us the importance of the nervous system in cutaneous pathology.

RESPIRATORY SYSTEM.—There is occasionally seen a close relationship between asthma and certain dermatoses, especially eczema, urticaria, and psoriasis. Psoriasis has been noted to appear and disappear with asthma, and in other cases has alternated with it. The explanation of this association of asthma with dermatoses is probably found in the fact that both are expressions of the same nervous instability.

SEXUAL SYSTEM.—There is a popular tendency, which is hardly warranted by the facts, to attach great importance to sexual disturbances as reflex causes of dermatoses. During adolescence disturbances of the sebaceous glands with seborrhea, comedones, and acne, are very common, but this is the expression of the greatly increased physiological activity of the appendages of the skin rather than of any pathological process arising in the sexual organs. Urethral irritation in the male and disordered menstruation are sometimes assumed as the cause of acne; but there is room for doubt as to any causal relationship between the conditions, and it is not improbable that the association is accidental or due to underlying causes which account for both conditions. Certainly treatment of acne based upon the relief of these sexual disorders *per se* does not give results that warrant any definite conclusions. Such vasomotor disturbances as erythema, urticaria, and herpes, are at times the result of reflex irritation from the sexual organs. The association of menstruation[1] with nervous disorders of the skin is closer, apparently, than that of sexual irritation in the male. It is not uncommon to see the recurrence with menstruation of herpes, either of the face or of the genitals, and it is very common to see an exacerbation of acne or eczema with menstruation. Rarely the association of bullous and pemphigoid lesions with menstrual disorders has been observed. The association of pregnancy with dermatoses is sometimes quite direct. The commonest lesion of the skin in pregnancy is the increased deposit of pigment which is seen about the face. Grave dermatoses, such as scarlatiniform erythema, impetigo herpetiformis, and dermatitis herpetiformis are in very rare instances found associated with it. It is not infrequent to see an existing disease of the skin altered by pregnancy. Thus eczemas and psoriasis may occasionally improve or disappear during pregnancy, or, on the other hand, they may be exaggerated. During the period of nervous instability which frequently characterizes the menopause, we see the same vasomotor disturbances that are referred to above in association with menstruation.

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[1] Bulkley, "Menstruation and Skin Diseases," The Rebman Co., 1906.

DENTITION.—The gastrointestinal troubles and the nervous irritability which occur with dentition are frequently associated with eczema and urticaria, and rarely with erythemas. But dentition alone does not play a rôle of any importance in dermatoses.

External Causes.—The external causes of cutaneous diseases are, of course, as numerous as the external irritants to which the skin may be subjected. For purpose of classification they may be divided into: (1) mechanical; (2) chemical; (3) actinic; (4) bacterial or parasitic.

MECHANICAL CAUSES.—The influence of mechanical causes in producing lesions of the skin we see in the innumerable lesions that result from friction, or pressure or other forms of external force, and such obvious causes need no enumeration.

Scratching.—The most important mechanical factor in diseases of the skin is the secondary effect of scratching. The sensation produced by scratching temporarily relieves itching, so that itching and scratching are constantly associated. The association is so intimate that it may be stated as a law that, unless voluntarily restrained, scratching always varies in vigor directly with the intensity of itching. Where itching is moderate, scratching will be carried only to the point of producing a hyperæmia or a moderate inflammatory reaction; where itching is intense, scratching will be carried to the point of producing excoriations and dermatitis. As the result of the varying degrees of traumatism from scratching we see varying manifestations of dermatitis. Where the itching and the scratching are temporary there will result only an evanescent dermatitis, which quickly subsides and leaves no traces. If, however, the two are persistent over weeks or months, the skin may become thickened, indurated, and pigmented as a result of a chronic dermatitis. In acute, violently itching dermatoses, such as acute eczemas, the essential pathological processes may be greatly exaggerated as a result of the traumatism which occurs from scratching. In all dermatoses where scratching is carried to the point of producing excoriations, the results of the mechanical traumatism are complicated by secondary infections from the nails, and thus in such conditions we get a secondary pustular dermatitis. The best illustration of this is seen in scabies, in which the typical condition is a pustular dermatitis that is more largely the result of traumatism and secondary infection from scratching than of the actual ravages of the parasite itself.

CHEMICAL CAUSES.—Chemical irritants may be so strong that momentary contact is sufficient to produce lesions of the skin varying in character, according to the intensity of the agent, from superficial dermatitis to destruction of the skin. Among such agents are carbolic acid, croton oil, the mineral acids, and the caustic alkalies. Other substances which are less active require more or less prolonged contact.

Such are the innumerable more or less irritating substances to which the skin is occasionally or habitually exposed. Any enumeration of them is impossible. They range from the ubiquitous dirt and soap and water to the many irritating substances to which workers in the different trades and arts are exposed. The lesions which these moderately irritating substances produce are usually varying degrees of dermatitis, with which may be combined secondary lesions, like excoriations, fissures, and the thickening of the skin which results from long-continued moderate dermatitis. In this class belong most of the irritants which produce the occupation dermatoses.

Among the commonest irritants of this class must be mentioned soap and water. We see their effects in the eczemas of washerwomen and janitors, whose hands are habitually exposed to the effects of water and strongly alkaline soaps. We see similar effects occasionally from the exaggerated use in the toilet of mild soaps and water by individuals with delicate skins.

Another class of chemical irritants which frequently produce dermatoses are decomposing body secretions. The fatty acids and other products of decomposition are irritating, and occasionally are the causes of dermatitis. An illustration of this is seen at times in dermatitis about the genitals of infants from contact with decomposing urine, or under pendulous breasts from the decomposition of sweat and sebum. In the eczema sometimes seen about the genitals of diabetics we have an illustration of a dermatitis due, not to an excretion which has become irritating from decomposition, but to one which from the sugar in it is primarily irritating.

In addition to the many irritating substances which have a more or less universal effect upon individuals, there are numerous substances which are irritating to individuals of particular sensibility. One of the commonest illustrations of agents of this sort is iodoform, which in susceptible persons may even in very small quantities cause intense and widespread dermatitis. The dermatoses produced by poisonous plants, like poison ivy, belong, for the most part, in this class.

ACTINIC CAUSES.—The effects which are produced by what may be called actinic causes are seen in lesions of the skin that result from heat, light, x-rays, and other forms of radiant energy. Aside from ordinary burns, heat is not a prolific cause of cutaneous lesions. We see occasional attacks of summer dermatitis which are apparently produced by heat independent of perspiration, but these are not common. There is occasionally seen in firemen and other persons, who are for any reason habitually exposed to radiant heat from a source of high intensity, a chronic erythema or dermatitis with much pigmentation on the exposed surfaces. The varying degrees of inflammation or destruction of tissues which result from freezing are the commonest manifestations of the effect of cold upon the skin. As a permanent result of freezing, and at times as a result of habitual exposure of the extremities to moderate degrees of cold, we occasionally see vasomotor disturbances, like sluggish peripheral circulation or erythematous lupus, in which there has been a permanent alteration in the vascular tone of the affected parts. The effect of sunlight is seen familiarly in the dermatitis which we call sunburn, and in the increase of pigment, which shows as tanning or freckling. These lesions are not due to heat, as is shown by the fact that they may occur in a low temperature, but they are directly the result of the effect upon the cells of the skin of the actinic rays at the violet end of the solar spectrum. The action of x-rays upon the skin is analogous to, but more intense than that of light. Where their effect is moderate, the results are dermatitis and tanning and freckling indistinguishable from those produced by light; where it is intense it may cause complete destruction of the tissues.

PARASITIC CAUSES.[1]—The microörganisms which are found in the skin are classified after Flügge into: (1) Streptotricheæ; (2) blastomycetes or yeasts; (3) hyphomycetes or molds; (4) schizomycetes, fission fungi, bacteria.

Streptotricheæ.—Streptotricheæ are the recognized pathogenic agents in two conditions which occur in the skin, actinomycosis due to the ray fungus or actinomyces, and madura foot due to the streptothrix maduræ. Other streptotricheæ have been found in lesions in the skin in a few rare cases.

Blastomycetes or Yeasts.—The blastomycetes of Gilchrist and Stokes has been demonstrated to be the pathogenic agent of blastomycetic dermatitis or blastomycosis. Another yeast, the saccharomyces subcutaneus tumefaciens, was apparently the pathogenic agent of a myxomatous tumor of the skin which was studied by Curtis. These are the only two conditions in which Blastomycetes are known to be the pathogenic agents. The so-called cancer bodies which have been described by Plimmer, Sanfelice, and others are, perhaps, varieties of yeasts, but concerning their essential relationship to carcinoma or indeed concerning their morphology, we are not at present in a position to make definite statements.

Hyphomycetes or Molds.—To these are due a number of well-recognized and common dermatoses. In all of these the pathogenic organisms are characterized by a tendency to grow in the horn substance of the skin, either of the epidermis or of the hair or nails, and the resulting dermatoses all present certain general resemblances. The commoner dermatoses produced by hyphomycetes are erythrasma, due to the microsporon minutissimum; pityriasis versicolor, due to the microsporon furfur; favus, due to the achorion schönleinii; and the different varieties of ringworm which are produced by several varieties of hyphomycetes. In man these are chiefly three: the microsporon audouini, the tricophyton endothrix, and the tricophyton ectothrix.

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[1] For detailed account of the bacteria of the skin reference must be made to the works on bacteriology or to the excellent chapters on this subject in MacLeod, to whom the author is greatly indebted.

Schizomycetes or Bacteria.—The influence of bacteria in cutaneous diseases is ubiquitous and extremely important. Unfortunately, however, the bacteriology of the normal as well as of the diseased skin is exceedingly complex, and our definite knowledge is correspondingly limited. In the first place, the healthy skin is the normal habitat of numerous bacteria. Tommasoli has described ten cocci, six bacilli, and six yeasts which may occur in any scale from the epidermis. Our knowledge of the morphology of the bacteria of the normal skin and of their possible virulence under certain conditions is far from complete, and this fact complicates the consideration of the bacteriology of almost every cutaneous disease. The most important of the bacteria of the normal skin is the staphylococcus epidermidis albus of Welch. This differs from the staphylococcus pyogenes albus chiefly in that it liquefies gelatin and coagulates milk less rapidly than that organism, and most bacteriologists, perhaps, believe that it is the staphylococcus pyogenes albus in a state of lower virulence. It would be a useless expenditure of time even to enumerate the organisms which have been suggested upon more or less plausible grounds as the pathogenic agents of different dermatoses. Any brief references to these must be taken up in considering the various diseases in connection with which the suggestions have been made. In a general way it may be said that the bacteriology of cutaneous diseases varies from definitely established knowledge, which meets all the requirements of the postulates of Koch, in some diseases to the merest suggestions in others. Thus we know the pathogenic bacteria of erysipelas, leprosy, tuberculosis, rhinoscleroma, anthrax, and glanders. In other diseases, like acne, chancroid, impetigo, and seborrheic dermatitis, organisms have been described which perhaps are the pathogenic agents, but positive proof is wanting. In other conditions, like alopecia areata, dysidrosis, erythema nodosum, and mycosis fungoides, various organisms have been described; but of the bacteriology of these conditions—even whether many of them are of bacterial origin or not—we really have no definite knowledge.

The most important pathogenic organisms in cutaneous diseases are the common microörganisms of suppuration. The staphylococcus pyogenes aureus, the staphylococcus pyogenes albus, the staphylococcus pyogenes citreus, and the streptococcus pyogenes, are likely to complicate any lesion of the skin in which the horny epidermis is destroyed and we consequently have to take into consideration their action in dermatoses. Usually, however, they only modify conditions which are th result of other essential causes. In certain conditions, however, as furunculosis and carbunculosis and sycosis, their rôle is of chief, if not sol importance. It is also not disproven that they are the active bacteri agents in certain of the most common pustular affections of the skir like acne, and impetigo and chancroid, in which attempts have been mad to establish other organisms.

Animal Parasites.—The skin is subject to the invasion of numerou animal parasites. These may be taken up more conveniently with tl consideration of the diseases which they produce.

IV.—PATHOLOGY [1]

The skin, embracing as it does so comprehensive a number of co stituent tissues, exhibits at one time or another almost all of the path logical conditions to which the body is subject—anæmias, congestio œdemas, inflammations, hypertrophies, atrophies, degenerations, a new growths of its various tissues. Cutaneous pathology, therefore, i volves almost the entire domain of human pathology. We have to co sider here only the characteristic lesions of the skin and such modifi tions of the various pathological processes as characterize their locati in the skin.

The skin presents a unique advantage for the study of pathology that the diseased tissues are open to direct inspection during life. Tl fact has added greatly to the importance of cutaneous pathology as contributor to general pathology.

Inflammation.—As in all pathology, inflammation is the most i portant process in diseases of the skin. Inflammation as it occurs in t corium does not differ from inflammation as it is seen in other vascul structures. There is at first hyperæmia with exudation of serum, d pedesis of leucocytes and erythrocytes, and swelling of the protoplasm the fixed tissue cells. As the process becomes more intense all of th phenomena are exaggerated, and proliferation of the true connecti tissue cells and of the cells of the walls of the blood vessels occurs. T connective-tissue bundles of the stroma are pressed apart by the infilt tion and may disintegrate and be absorbed. In a like manner and mc quickly the elastic tissue is affected and breaks down and is absorb

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[1] MacLeod.—Darier, "La Pratique Dermatologique."—Auspitz, *v. Ziemsse Handbook of Skin Diseases.*—Unna's "Histopathology."—Adami, *Allbutt's Syst of Medicine.*—Hektoen, "International Text Book of Pathology."—Crocker.

It might be added, too, that when repair takes place the restitution of the elastic tissue is slower and less complete, so that in scar tissue any regular elastic network is apt to be lacking. In the blood vessels there is seen every degree of nutritive change from simple swelling of the endothelial cells to extreme peri- and endarteritis and phlebitis.

The termination of this process is various. If the reaction is of slight degree and temporary, the inflammatory exudate may be absorbed and complete restitution occur quickly. If the process is long continued the cellular infiltration becomes dense and the proliferation of connective-tissue cells abundant, and when the reaction subsides restitution to normal is comparatively slow. When the reaction passes the bounds of reparative and defensive activity it may defeat its own purposes, and result in such interference with the nutrition of the parts that there occurs not only degeneration of the cellular exudate, but destruction of the fixed stroma, producing suppuration or necrosis. Whenever these occur, the removal of the pus and of the fibrin deposited in the tissues and of the necrotic masses renders the reparative process much slower.

In the removal of the products of inflammation the most important factor is the leucocytes. Those which are destroyed liberate ferments which digest the fibrin and the other inflammatory elements, while those which remain active serve as the chief vehicle for the removal of the inflammatory products.

Whenever there is destruction of the stroma of the corium this must be repaired by the formation of new connective tissue. If this destruction involves the surface of the skin we have ulceration, and repair takes place by the formation of connective tissue and the production of scars. We have also in a similar way the formation of permanent connective tissue, where there is destruction of the stroma without ulceration, as seen frequently after the lesions of lupus, lupus erythematosus, and syphilis. The condition is analogous to that of interstitial sclerosis, and results in the formation of scars without the occurrence of previous ulceration. In long-continued inflammatory reactions of moderate intensity in the skin true interstitial sclerosis may occur with the formation of a large amount of permanent fibrous tissue. This is seen especially in dependent parts after long-continued moderate dermatitis, or where there is lymphatic obstruction to interfere with the absorption of the inflammatory exudate. The formation of young connective tissue is abundant in many inflammatory dermatoses. In these conditions, however, the new tissue does not become permanent, and, as the reaction subsides, gradually disappears.

The cellular infiltration in inflammation of the skin includes the various types of cells that are usually found in inflammation—erythrocytes in greater or less abundance, polymorpho- and mononuclear leucocytes, young connective-tissue cells, and, in varying numbers and under varying conditions, eosinophiles, plasma cells, mast cells, and giant cells. For descriptions of these reference must be made to works on pathology.

The location of an inflammatory reaction in the corium depends upon the site of irritation. When the irritant is external the first changes are found around the blood vessels of the papillary and subpapillary layers. When it is in the corium itself the first changes occur in the neighboring blood vessels. Most frequently inflammation begins around the follicles of the skin, doubtless for the reason that these are the most vulnerable parts and are likely, therefore, first to be affected by injurious agents. Diffuse cellular infiltration of the corium rarely occurs.

Epidermal Inflammation.—As the epidermis is a nonvascular structure, inflammation in it cannot be dissociated from inflammation in the underlying corium from which it obtains its nutrition. The first step in inflammation of the epidermis is effusion of serum and wandering of leucocytes and of a few erythrocytes into the inter-epithelial spaces of the mucous layer. As a result of the œdema these spaces become distended and the mucous cells become swollen, with an increase of the perinuclear spaces. The nutrition of the cells is interfered with; there is an increased formation of pigment and a rapid proliferation of imperfect cells, which in their evolution remain more or less moist and do not become perfectly cornified. If the inflammatory reaction is intense the epithelial cells rupture or disintegrate, the horn cells become macerated, and vesicles, bullæ, pustules, or areas denuded of horny epidermis develop. The formation of these various lesions, together with the distrophies which occur as a result of inflammation of the epidermis, will be considered in greater detail when we take up the consideration of the characteristic lesions of the epidermis.

The clinical expression of inflammation of the skin is easily explained by the histological changes. There is first redness, varying in intensity with the hyperæmia which produces it. As a result of the nutritive changes in the epithelial cells the unbroken epidermis in the affected areas has a hazy, ground-glass appearance. The increased proliferation of epithelium and the incomplete cornification produce increased desquamation. There is more or less swelling as a result of the inflammatory exudate. If this is diffuse, which is rarely the case, the swelling is likewise diffuse. Usually the inflammatory exudate is circumscribed, particularly around the follicles, and these circumscribed infiltrations produce roundish, more or less conical elevations of the skin which are solid or contain free fluid, according as the tissue is intact or has ruptured under the tension of the infiltration. If the horny epidermis has been destroyed, there is exposed a thin layer of succulent mucous cells which show as a blood-red surface from which escape of serum is unchecked, and upon which a deposit of fibrin and disintegrated cells takes place. When an inflammatory reaction is chronic in intensity and persistent in course, the dense inflammatory infiltration shows clinically in thickening and induration of the corium which vary greatly according to the extent of the infiltration. At the same time there is likely to be hypertrophy of the epidermis with the formation of a thickened but imperfect horny layer. These various changes in the corium and epidermis result in a thickened, hard, scaly skin of diminished elasticity and of greatly impaired function.

Congestion, œdema, and, to a certain extent, hæmorrhage, are all features of inflammation. But in certain lesions of the skin each of these processes is the salient characteristic, the other features of the inflammatory process being overshadowed and relatively insignificant, so that these conditions merit brief separate consideration.

Congestion.—Transient hyperæmia of reflex vasomotor origin may be a physiological process, the chief illustration of which is seen in emotional flushing of the face. Such evanescent hyperæmia is probably entirely independent of any inflammatory changes. Even these physiological hyperæmias, however, if for any reason they become persistent, easily pass over into actual inflammatory processes, as is seen in the inflammatory changes in the flush area of the face which occur as a result of persistent congestion.

Aside from transient physiological flushing, the active congestions of the skin represent simply the first stages of an inflammatory reaction with the essential changes which have been referred to under slight inflammation of the skin.

Passive congestion is usually of mechanical origin, but in certain conditions it is due to vasomotor disturbance. It may be accompanied by inflammation or it may occur without it. If unaccompanied by inflammation there are no histological changes except those of slight œdema. When associated, as it usually is, with inflammatory changes, there is likely to be an unusual degree of hyperplasia of connective tissue and of increase in the production of pigment.

According to the intensity of active hyperæmia the color of the skin varies from a pinkish to a bright red; in passive hyperæmia the skin is cold and of violaceous color. In both active and passive congestion the color disappears on pressure, owing to the fact that under pressure the blood, which causes the color and is contained in the vessels, is driven out. In contrast to this, lesions produced by pigment or extravasated blood free in the tissues do not lose their color under pressure. Active hyperæmia may be diffuse or it may occur in spots or small patches of varying sizes. True diffuse erythema is uncommon; even eruptions that give the appearance of diffuse erythema are usually made up of closely set punctate lesions which more or less completely coalesce. Passive hyperæmia usually occurs in diffuse areas.

Anæmia.—Generalized cutaneous anæmia is produced by general constriction of the cutaneous blood vessels. This is seen in several conditions. It may occur after severe hæmorrhage, in which case the anæmia is partly due to the actual diminution in the quantity of blood, but is in larger part due to a purposeful reflex stimulation of the vasoconstrictor centers of the cutaneous blood vessels in order that the tissues of greater organic importance may have a larger blood supply. Psychical disturbances and shock may also cause cutaneous anæmia, and it may also take place as a result of toxins circulating in the blood and stimulating the vasoconstrictor centers (MacLeod). Local cutaneous anæmias are produced by local interference with cutaneous circulation. A permanent vasomotor disturbance of this sort may develop as the result of the disturbance of the circulation produced by frostbites. A similar cutaneous anæmia is seen in the profound disturbance called Raynaud's disease. Localized anæmia also results from mechanical obstruction of the blood flow. The best illustration of this is the evanescent, circumscribed anæmia which is often produced by the intense œdema of wheals. It may also be produced by an embolus or thrombus, by an obliterating endarteritis, or by a periarteritis. Except as it occurs in wheals, circumscribed anæmia is not a common lesion of the skin.

Clinically cutaneous anæmia shows as pallor and coldness of the skin. Cutaneous anæmias due to changes in the quality of the blood are only parts of general systemic conditions. They are seen in tuberculosis, syphilis, malignant growths, leukæmia, and various other organic diseases. The pallor of the various cachexias is not of the same origin as that of the cutaneous anæmias due to diminution in the quantity of blood in the skin, for in these conditions the deficiency is not in the quantity of the blood but in its quality.

Œdema.—Œdema of the skin, like œdemas in general, is of mechanical or of vasomotor origin. In mechanical œdema there is transudation of serum, poor in albumin and leucocytes, and there are usually no demonstrable vascular changes. The lymphatic and interfasicular spaces of the corium and the intercellular spaces of the mucous layer of the epidermis are distended. If the condition is persistent the connective-tissue fibers separate and undergo degenerative changes and the elastic fibers disintegrate and are absorbed. Vasomotor œdema may be regarded as an aberrant inflammatory process. There are the usual changes of inflammation, but the exudation of serum is the dominant feature. Vasomotor œdema of the skin may be only a part of an extensive œdema, involving all of the tissues of the part, but it also occurs as a distinctly cutaneous lesion. In cutaneous vasomotor œdema the œdema is most pronounced in the papillary and subpapillary parts of the corium, and extends upward into the inter-epithelial spaces of the superjacent mucous layer. Localized cutaneous œdema produces characteristic lesions called wheals, for description of which see urticaria.

Hæmorrhage.—In all the inflammatory processes there is a greater or less exudation of red cells. In certain violent types of inflammation of the skin, as zoster and variola, extravasation of red cells may occur in large numbers and produce hæmorrhagic lesions. In these conditions the extravasation of red cells is apparently an exaggeration of the ordinary diapedesis of red cells seen in inflammatory processes generally. In addition, however, to this inflammatory hæmorrhage into the skin there occur true hæmorrhages into the skin (purpuras) in various systemic disturbances. The blood as a whole, with white and red corpuscles, infiltrates the corium to a variable depth. In such lesions there is a deposit of fibrin and abundant extravasation of red and white cells free in the tissues. The lesions usually disappear without damage to the skin, but in certain violent purpuras gangrene of the tissues may occur.

Clinically hæmorrhage into the skin produces lesions which at first are dark red to blackish from the presence of deoxygenated blood; then from the liberation of hæmatin in the disintegration of the erythrocytes the lesions become brownish purple, then greenish, then yellowish, and finally gradually disappear; in other words, the lesions go through the same involution as an extravasation of blood in the skin from a traumatism. The pathological changes which underlie these lesions are not clearly made out. They may occur from diminished perivascular tension, as illustrated by the extravasation of blood into the skin which occurs in high altitudes. In Sack's investigations by means of serial sections, he was able to discover a rupture of the vessel walls in certain hæmorrhagic lesions. Cheyne found bacilli blocking the small arteries in lesions which he examined. By others alterations in the vessel walls of various sorts have been invoked as an explanation, and again changes in the blood. That these lesions are essentially of vasomotor origin in many cases is shown by their frequent association with other lesions of inflammatory or œdematous character.

Vesicles, Bullæ, Pustules.[1]—The mechanism of the formation of vesicles, bullæ, and pustules is essentially the same.

Vesicles.—Vesicles are produced through the pressure of serum in the epidermis causing either separation or rupture of the cells, thus forming cavities in the epidermis which are filled with serum. Two factors, therefore, enter into the formation of vesicles: (1) the pressure of the fluid in the tissues, and (2) the strength of the union between the cells or the strength of the cells.

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[1] For definitions, see elementary lesions of the skin.

In certain rare conditions of the epidermis the formation of vesicles is primarily due to a lack of normal strength of the prickle cells and their intercellular connections, so that slight increase in pressure of the lymph in the intercellular spaces of the mucous layer results in rupture of the intercellular connections and the formation of cavities. Such vesicles may form without the appearance of congestive phenomena. To this rare condition Auspitz gave the name acantholysis. Of very much more frequent occurrence is the formation of vesicles under conditions of exaggerated tension of the fluid in the epidermis. In such conditions both factors in the production of vesicles are active. There is a reduction in the strength of the cells as a result of the œdema, so that they give way more readily; but an equally important, or more important, factor is the increased tension of the serum, which causes rupture of the intercellular connections or of the cells themselves. Weidenfeld has shown that bullæ can be produced by the pressure factor alone.

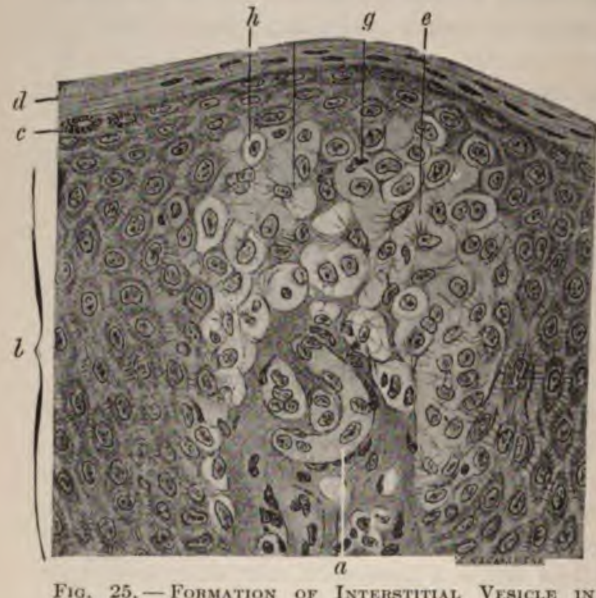

FIG. 25. — FORMATION OF INTERSTITIAL VESICLE IN CHRONIC ECZEMA. *a*, Apex of a papilla; *b*, stratum mucosum; *c*, stratum granulosum; *d*, stratum corneum; *e*, intercellular spaces filled with clear serous fluid; *g*, leucocytes; *h*, cell showing ballooning degeneration. (Darier.)

In the formation of vesicles the mechanism may consist of rupture of the intercellular processes or of rupture of the cells themselves, and, according to the mechanism of their production, Darier has divided vesicles into interstitial and parenchymatous. This division of vesicles is in great part for convenience of study and description, for, as a matter of fact, both intercellular rupture and rupture of the cells themselves occur in the ordinary process of vesiculation, but, according to variations in the process the formation of the lesions may tend toward one method or the other. The typical parenchymatous vesicle is found in variola and vaccinia; interstitial vesicles are those of eczema, zoster, scabies, dermatitis herpetiformis, and most inflammatory dermatoses.

In the formation of a vesicle there first occurs an outpouring of serum and wandering of a few leucocytes into the lymphatic spaces of the epidermis. With this there develops œdema of the cells, and as the reaction progresses and the hydrostatic pressure increases, intercellular connections break, cells rupture, and minute cavities filled with serum appear in the epidermis. The cells around the cavities are œdematous and swollen, or perhaps spindle-shaped from pressure. Larger vesicles are formed ordinarily by the rupture of the intervening walls of these minute vesicles. The larger vesicles thus formed may be unilocular, or more rarely multilocular, with strands of unbroken cells crossing them.

The location of vesicles may be in any part of the epidermis. The rupture of the cells, of course, occurs at the point of least resistance within the area of pressure. In œdema of the epidermis this is ordinarily in the upper part of the mucous layer or at the juncture with the granular layer; so that if the œdema is diffuse the formation of the vesicle occurs at this site. If, however, the outpouring of serum from the corium into the epidermis is intense and sudden there may be vio-

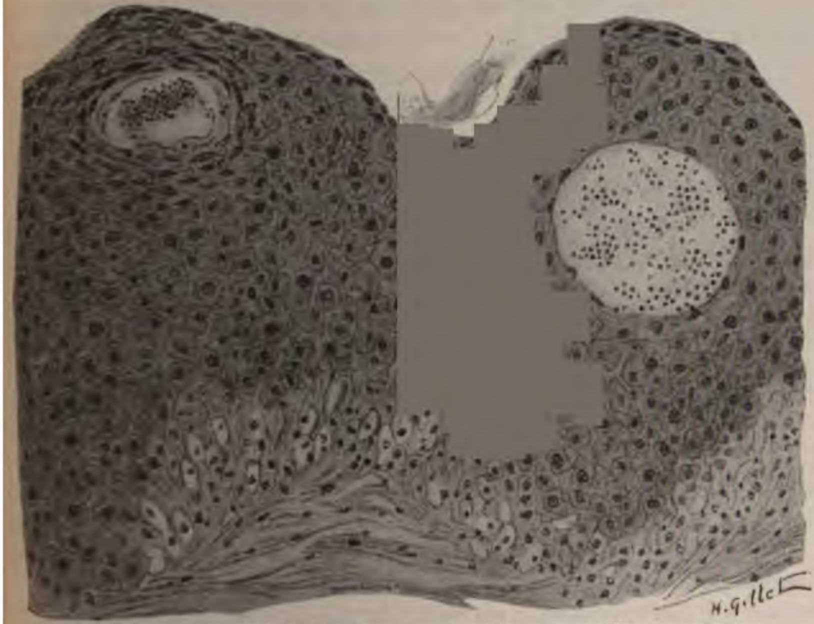

FIG. 26.—INTERCELLULAR VESICULATION IN DERMATITIS HERPETIFORMIS. (Leredde.)

lent tearing of the cells by the flood of serum, with the formation of the vesicle in the lowest parts of the mucous layer and with uplifting of the entire epidermis. If the vesicle has formed in the upper part of the mucous layer, and if after its formation the œdema has diminished to some degree, the epithelial cells of the floor of the vesicle may become cornified and the vesicle thus be encased and uplifted intact into the horny layer.

The contents of vesicles consist of coagulable plasma, a few leucocytes and degenerating epithelial cells, and epithelial nuclei.

At the bottom of vesicles of a certain type (zoster and erythema multiforme) and in their neighborhood, swollen round cells may be observed, which, from their resemblance to sporozoa have been described as organisms. Unna has shown that these are degenerating epithelial cells, and has given them the name "balloon" cells. These have lost their prickles by rupture, and if not under pressure are round or globular from œdema. They are, however, plastic and under pressure may assume any shape. The differentiation of the protoplasm from degeneration has been lost, and it has become a homogeneous mass which stains red with acid dyes. The nuclei, showing the characteristics of other nuclei in having greater resistance, have not degenerated, stain red, and are capable of division so that the cells may become multinuclear, like giant cells. As the œdema increases they gradually become more and more swollen and finally break down.

A vesicle may terminate in one of three ways: (1) If the reaction subsides with the formation of the vesicle, its contents may be absorbed without rupture of its walls. In that case cornification takes place regularly in the cells of the floor of the vesicle, and the lesion ends by the exfoliation of its roof without destruction of continuity of the horny layer. (2) If the causative process continues the roof of the vesicle may rupture. In that case the mucous layer is exposed and shows as a small bright red spot from which are exuded serum and other inflammatory elements, which dry upon the surface in the form of a translucent layer composed chiefly of fibrin. (3) The vesicle may develop into a pustule.

Pustules.—In all vesicles a certain number of leucocytes are present, their number increasing with the age of the vesicle; for the vesicle to become a pustule it is only necessary for the leucocytes to become abundant and undergo degeneration. The reason for the difference in the character of the contents of the various grades of lesions between vesicles and pustules has been accounted for by the French school by assuming different varieties of chemotaxis. They suggest, for example, a chemotaxis that attracts serum, a second which attracts serofibrinous fluid, and a third, the ordinary form of positive chemotaxis, which attracts serum and leucocytes. Accordingly, certain lesions persist as vesicles; others, on the other hand, according to the French—and their contention is generally accepted now—are pustules from the start—(*pustule d'emblée*). It is very rare, however, for a pustule not to begin as a vesicle, and, on the other hand, a vesicle if persistent usually becomes a pustule from invasion of leucocytes.

The invasion of leucocytes into pustules may be produced by any positive chemotactic agent. This is most frequently the toxin of one of the common pyogenic organisms. In variola and vaccinia pustules may develop without secondary infection with the common pyogenic organisms, their production being doubtless due to the specific organisms of the disease. Pustules may also be produced without infection by certain irritating chemicals, like mercury and croton oil.

In addition to the epidermic pustules which we have been considering, deep or dermic pustules also occur. These are found in suppuration of the follicles of the skin and are truly minute abscesses, presenting all of the usual characteristics of suppuration in connective tissue.

Bullæ.—The formation of bullæ is similar to that of vesicles. Like vesicles they occur in any part of the epidermis below the horny layer. Their site is most frequent, perhaps, between the horny layer and the mucous layer, but when the effusion of serum is sudden or when the overlying epidermis, like that of the palms and soles, is very resistant, the entire epidermis may be uplifted and the bullæ form between the epidermis and the papillary layer. In the formation of bullæ from intense and suddenly applied heat a peculiar process may occur. Here we have from the intense heat a sudden development of steam which violently ruptures the epidermis with the formation of cavities that readily fill with serum. Except under these conditions the bullæ in burns are of the usual inflammatory type.

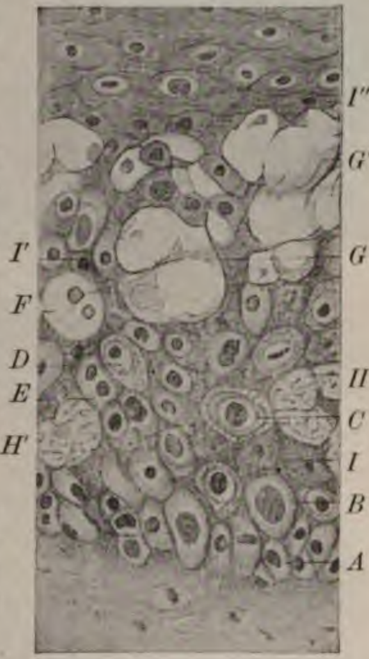

Fig. 27.—Parenchymatous Vesiculation in a Variolous Pustule. *A*, Cells of basal layer showing various degrees of alteration; *B*, cell with hypertrophic nucleus surrounded by a clear space; *C*, cell showing a clear perinuclear space, surrounded by a layer of protoplasm with the prickles persisting; *D*, dilatation of perinuclear space; *E*, cell with two nuclei; *F*, two cells, the cavities of which are separated by a thin membrane; *G*, *G′*, vesicular cavities due probably to the confluence of several cellular cavities; *H*, *H′*, cellular spaces filled with a fibrinous network; *I*, *I′*, *I″*, cells which have escaped the parenchymatous vesiculation. (Darier.)

The contents of bullæ, like those of vesicles, at first consist of serum, epithelial *débris*, and a few blood corpuscles. Bullæ may be sterile at first, but they quickly become contaminated with the organisms of the epidermis. As leucocytes invade them and degenerate, their contents become pustular. The production of the purulent contents of bullæ is thus accounted for in the same way as that of pustules. In addition, however, Darier has shown that the puriform contents of the bullæ of pemphigus foliaceus may be produced by the presence in the serum, not of ordinary pus cells (degenerated leucocytes), but of degenerated epithelial cells.

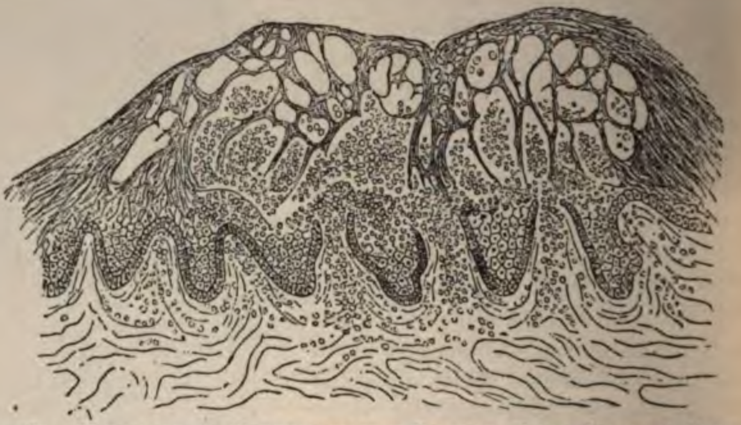

FIG. 27*a*.—MULTILOCULAR, PARENCHYMATOUS PUSTULE, EARLY STAGE, IN VARIOLA. (Rindfleisch.)

Hypertrophy of the Mucous Layer: Acanthosis.—In addition to the inflammatory changes in the mucous layer which we have been considering, the layer may undergo a true hypertrophy, in contradistinction to a malignant proliferation, as a result of irritation produced by pathogenic agents which are incapable of causing degeneration of the mucous cells or violent inflammatory infiltration. To this true hypertrophy of the mucous layer Auspitz gave the name hyperacanthosis (from ἀκάνθα, a prickle), and Unna later suggested for these lesions the term acanthoma and for the condition acanthosis. In acanthosis there is a proliferation of typical prickle cells; the basal layer is preserved intact and, as in the normal epidermis, limits the proliferation of the cells. In acanthosis, however, the proliferation of the cells is not confined, as normally, to the basal layer, but the cells divide and mitotic figures may be observed for several rows above the basal layer. The mucous layer may become several times its normal thickness. This increase in the mucous layer may be accompanied or unaccompanied by increase in the horny layer.

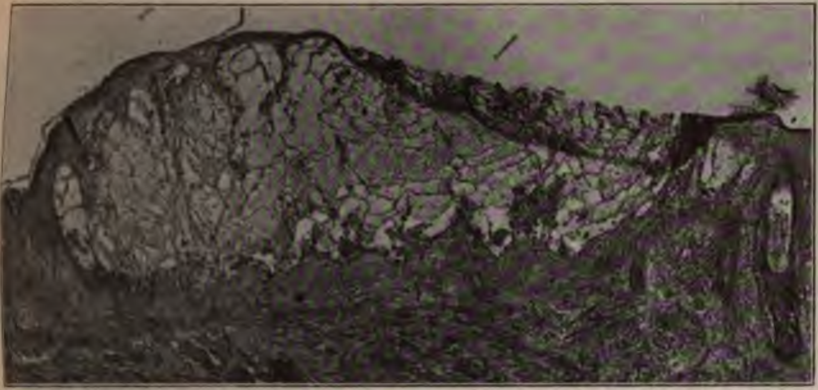

FIG. 1.—MULTILOCULAR VESICLE ON VARICELLA. × 50. (Harris's preparation.)

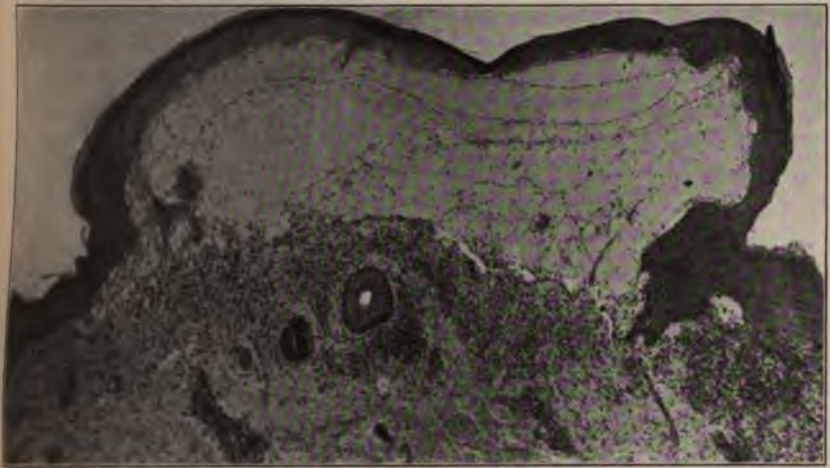

FIG. 2.—BULLA IN LICHEN PLANUS. Entire epidermis raised. × 48. (G. F. Harris's preparation.)

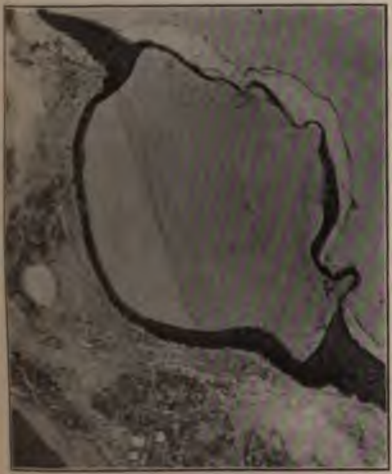

FIG. 3.—BULLA BETWEEN THE LAYERS OF THE EPIDERMIS, FROM ŒDEMA. (Author's collection.)

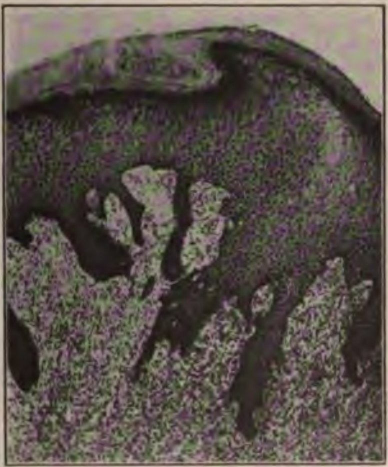

FIG. 4.—SKIN AT BORDER OF ULCER OF LEG. Showing inflammatory infiltration of corium, and with œdema and acanthosis and hyperkeratosis of epidermis. × 80. (Author's collection.)

As a result of the downward pressure of the hypertrophied mucous layer, the interpapillary processes become exaggerated in depth and there is a corresponding exaggeration in the length of the papillæ of the corium. For this reason the term papilloma has been commonly applied to the lesions of acanthoma, but, as Auspitz and Unna have shown, the apparent hypertrophy of the papillæ is secondary and due, not to their actual upgrowth, but to the downgrowth between the papillæ of the proliferating epithelial cells. Acanthosis is usually accompanied by inflammatory changes in the corium. It is seen in connection with numerous inflammatory reactions and in certain affections without inflammatory phenomena. The process is usually circumscribed. Typical acanthomata are verrucæ and condylomata. The condition is seen in various inflammatory affections, like psoriasis, seborrhœic eczema, herpes zoster, lichen planus, in all of which it is secondary to inflammatory changes in the corium. Acanthosis also occurs in the skin lesions of the granulomata, syphilis, tuberculosis, sarcoma, blastomycosis, and mycosis fungoides.

Atrophy of the Mucous Layer.—Atrophy of the mucous layer is represented by a decrease in thickness of the layer and disappearance of the interpapillary epithelial processes. Where the condition is marked the layer is represented by a thin line of cells, the papillæ are almost obliterated, and the boundary between the epidermis and corium becomes almost straight. A greater or less degree of atrophy of the mucous layer occurs normally in the general atrophy of the skin which is characteristic of old age. This senile atrophy of the mucous layer is frequently accompanied by an increased production of pigment in the deeper cells and by circumscribed hypertrophies of the horny layer. An atrophy of the skin similar to senile atrophy may be produced from exposures to x-rays. In the rare condition known as diffuse idiopathic atrophy of the skin there is, in connection with the general atrophy of the skin, a corresponding atrophy of the mucous layer. Atrophy of the mucous layer results most frequently from mechanical causes. These may be external or internal pressure. As an example of the former type we have the atrophy resulting from the continuous wearing of bandages or of tightly constricting garments. A similar atrophy of the mucous layer from external pressure occurs under callosities and corns, from the downward pressure which they exert. Atrophy of the mucous layer may be produced by internal pressure of any sort that puts the skin under tension. Illustrations of atrophy of this type are seen in the skin over malignant growths, in ascites, in pregnancy, and in extensive accumulations of fat. The atrophic striæ or white lines which develop in pregnancy are typical illustrations of this condition.

Pseudo-Hypertrophy of the Mucous Layer: Carcinoma.—In malignant proliferation of the epidermis, carcinoma, we have, in contradistinction to true hypertrophy of the prickle cells, a pseudo-h trophy; that is, a proliferation of poorly differentiated epithelial which do not undergo the normal evolution into horn cells, but r only the function of indefinite proliferation. The cells revert t type of embryonal cells, they lose their prickles, they become r or oval, the basal layer disappears, and the cells invade the surrou tissues.

Alterations in the Stratum Corneum.—The stratum corne subject to various pathological alterations. It may become hypertro —hyperkeratosis; it may undergo atrophy; cornification of the cell be imperfect—parakeratosis, or the cells may undergo degene changes resulting in anomalies of cornification—dyskeratosis.

Hyperkeratosis.—The term hyperkeratosis is applied to those tions of the stratum corneum in which there is an increased thickn the horny layer with complete cornification of the cells. The ex tion of hyperkeratosis lies in the fact that the horn cells become resistant than usual, so that their desquamation is postponed layer thicker than normal accordingly accumulates. This inc resistance of the cells in hyperkeratosis is due to an excessive p tion of keratin. In extreme cases the cells become so completely co that they form dense, hard, almost homogeneous horny masses; in cases the hyperkeratosis shows simply as an increased accumulat ordinary horn cells. Hyperkeratosis may occur with hypertrop the mucous layer, but it may occur independent of any acanthosi the other hand acanthosis may occur with or without hyperker so that the two processes are essentially independent, hyperke being due to changes occurring independently in the cells stratum corneum. According to Darier hyperkeratosis is always ciated with increased thickness of the granular layer, but while usually the case, it does not always occur, and, indeed, in some the transitional layer may even be diminished (MacLeod). Th important factor in the production of hyperkeratosis is increased sure or friction upon the horn cells, to which increased demand their function as a protective structure they respond by the for of a more resistant quality or a greater quantity of keratin than n The most familiar examples of hyperkeratosis are the horny mass callosities and corns which develop upon exposed surfaces. Hyp tosis may be circumscribed or diffuse. It occurs in various patho conditions and in certain congenital anomalies; in these latter c may be universal.

Parakeratosis.—The term parakeratosis was suggested by A and has been accepted by usage to describe conditions of imperfect fication. In parakeratosis usually, but not invariably, there is i in thickness of the horny layer, but in contrast to hyperkeratosis the cells are imperfectly cornified and form friable scales. Parakeratosis occurs as the result of inflammatory changes in the mucous layer, and is always preceded by œdema of this layer, with dilatation of the interepithelial lymph spaces, swelling of the epithelial cells, and infiltration of the interepithelial spaces with leucocytes. The transitional layers are involved in the same process, and keratohyalin and eleidin are usually abundant. This condition of the deeper cells of the epidermis interferes with the normal evolution of the epithelial cells and their final form is rendered imperfect. The horn cells remain moist and somewhat swollen, their nuclei persist, and the formation of keratin is incomplete, so that spaces remain between the horn cells, and in these air, serum, and leucocytes are found. The cells are less resistant than normal and desquamation is therefore rapid. The lesions of psoriasis present a typical condition of parakeratosis. It occurs also in scaly eczemas, lichen planus, seborrhœic eczemas, and in various inflammatory diseases of the skin which are accompanied or followed by desquamation.

Anomalies of Cornification: Dyskeratosis.—In the words of Darier, who has, perhaps, done more work in this field than anyone else, " The name dyskeratosis is used to designate epithelial alterations in which a certain number of isolated Malpighian cells become differentiated from their neighbors, not progressing toward normal keratinization, but individually undergoing special morphological and chemical modifications; dyskeratosis may be considered as unicellular parakeratosis." These anomalous cells become partially cornified, but at the same time their protoplasm, instead of being converted into the usual waxy substance, undergoes what is apparently colloid or hyalin degeneration, the process resulting in peculiar bodies which have been regarded as sporozoa, but are now generally admitted to be anomalous horn cells. These cells are roundish, of the size of prickle cells, and have a granular protoplasm inclosed in a double-contoured membrane. Dyskeratosis occurs, but with variations, in several conditions: in molluscum contagiosum, forming the so-called molluscum bodies; in Darier's rare psorospermosis follicularis vegetans; in Paget's disease and other epitheliomata; and occasionally in horns and warts.

Atrophy of the Stratum Corneum.—Atrophy of the stratum corneum occurs under conditions similar to those producing atrophy of the stratum mucosum which we have considered. It also occurs at times in general diseases which interfere with the nutrition of the skin and in certain nervous diseases, like tabes dorsalis.

Pathology of the Corium.—The pathological processes which occur in the various constituent tissues of the corium present the usual features of these processes as they occur in other vascular tissues, and, as the corium is less characteristically a cutaneous structure, viewing the skin as an organ, than the epidermis, so its various pathological alterations show fewer peculiar modifications. The pathology of the corium is the pathology of connective-tissue and vascular structures in general, and for an intimate consideration of the various pathological alterations to which its constituent tissues are subject reference must be made to works upon general pathology.

Blood Changes in Diseases of the Skin.—Eosinophilia is the commonest change found in diseases of the skin. French[1] has shown, however, that eosinophilia is much less frequent than has been commonly supposed. He examined thoroughly ninety patients from the out-patient department of Guy's Hospital who were suffering from skin diseases. Assuming that it requires more than five per cent eosinophile cells to constitute eosinophilia, he found eosinophilia in only thirteen of his ninety patients, and in only four was the eosinophilia marked. His conclusions are as follows:

"In these ninety patients:

"(1) There were a very large number who showed no eosinophilia at all.

"(2) There were a few who showed slight eosinophilia.

"(3) There were only four who showed marked eosinophilia.

"(4) There were none of the skin conditions investigated in which eosinophilia was not the exception rather than the rule, except in (*a*) pemphigus, (*b*) dermatitis herpetiformis, and possibly in (*c*) xanthoma diabeticorum.

"(5) There were a large number of skin affections in which, as far as the research went, no single case of eosinophilia occurred.

"(6) There were parasitic skin diseases showing no eosinophilia.

"(7) There were a few individuals suffering from psoriasis, from eczema, or from syphilis who showed eosinophilia, but many who showed none, so that, owing to its erratic occurrence, the presence or absence of eosinophilia will not serve as a means of diagnosis between syphilitic affections and the other two.

"(8) There was no evidence in favor of 'Cannon's law.'

"(9) There was, upon the whole, but with individual exceptions, a relative diminution of polymorphonucleated cells.

"(10) There was a tendency to a relatively high proportion of lymphocytes.

"(11) The fall in percentage of polymorphonucleated cells was approximately equaled by the rise in percentage of small lymphocytes.

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[1] *Guy's Hospital Reports*, 1903; *Abs. Brit. Jour. Derm.*, 1904.

"(12) The lymphocytosis was not constant, except in the patients suffering from (*a*) congenital syphilis, (*b*) urticaria, in each of which conditions it was well marked."

V.—SYMPTOMATOLOGY

"Affections of the skin have the particular characteristic that they are visible. The lesions which they produce are for a large part directly perceptible, and when we examine them with the naked eye or with the lens we are working with macroscopic pathological anatomy. Thus the general symptomatology of the dermatoses differs completely from that of internal maladies, in which we must have recourse to methodical and minute examination and exploration of functional disturbances in order to deduce by reasoning the probable lesions" (Brocq). In respect to ease of direct examination dermatology has a unique advantage over other departments of medicine. The lesions of the disease which the dermatologist is studying are spread before him, and the objective symptoms are accordingly of paramount importance.

ELEMENTARY LESIONS

The innumerable clinical pictures which the dermatoses present are made up of relatively few elementary lesions, or, to use Besnier's words, essential primary eruptive elements. These elementary lesions are of two kinds, primary and secondary. The primary lesions are the original forms in which the various lesions appear; the secondary lesions are those which develop out of the primary, as result either of their further transformation or of adventitious agents acting upon them.

A clear comprehension of the elementary lesions of diseases of the skin is prerequisite to the study of dermatology, for it is in the terms of these lesions that dermatoses must be described. For this reason a favorite—and accurate—metaphor of dermatologists is to speak of the elementary lesions as the a b c of dermatology.

The primary elementary lesions are *macules, papules, vesicles, pustules, bullæ, tubercles, tumors,* and *wheals.* The secondary lesions are *scales, crusts, excoriations, fissures, ulcers, scars,* and *stains.* As will be seen on more minute examination, there are a few special lesions characteristic of peculiar conditions which must be added to the above, but, leaving out these unimportant exceptions, all dermatoses are expressed by various combinations of these elementary lesions.

Primary Lesions. Macules.—A macule is a circumscribed discoloration of the skin without elevation; in other words, a macule is a spot in the skin.

Papules.—A papule is a solid elevated pathological formation of the skin not larger than a split pea.

Vesicles.—A vesicle is an elevated[1] pathological formation of the skin, not larger than a split pea and containing free serous fluid.

Pustules.—A pustule differs from a vesicle in that it contains pus.

The order in which macules, papules, vesicles, and pustules are placed above is that in which transformation of these lesions from one to another naturally takes place. In many inflammatory conditions the first punctate hyperæmia of the inflammatory process shows as a macule; if the process becomes more intense, the exudate produces a minute swelling which shows as a papule; if it becomes still more intense, it may lift up the epidermis with the formation of a vesicle; and if it continues still further, invasion of the cavity of the vesicle with leucocytes and their subsequent degeneration produces a pustule. It is needless to say that lesions do not always go through this complete evolution.

Bullæ; Blebs.—A bulla or bleb is an elevation of the skin above the size of a split pea and containing free fluid.

Tubercles and Tumors.—A tubercle is conventionally described as a solid pathological formation of the skin between the size of a split pea and a hazelnut, and a tumor as a solid pathological formation of the skin above the size of a hazelnut. And the terms are so used in dermatology, but, as will be seen on more complete description, these definitions do not cover completely the meanings attached to these terms.

Wheals; Urticæ; Pomphi.—A wheal is a peculiar circumscribed elevation of the skin produced by œdema of the corium.

Secondary Lesions. **Scales; Squamæ.**—A scale is a mass of exfoliating or exfoliated epidermis.

Crusts.—A crust is a mass formed upon the surface of the skin from the accumulation of dried exudate or other *débris* of disease.

Excoriations.—An excoriation is a superficial abrasion of the skin.

Fissures or Rhagades.—A fissure is a crack in the skin usually extending through the epidermis into the corium.

Ulcers.—An ulcer is a circumscribed loss of substance of the skin extending through the epidermis into the corium and produced by disease.

Scars or Cicatrices.—A scar or a cicatrix is a connective-tissue formation which replaces a previous loss of substance of the corium.

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[1] Elevation is a part of the usual conception of papules and vesicles, but these terms are applied with accuracy to some lesions which are essentially of the character of papules and vesicles but are so imbedded in the skin that they are not elevated. The chief illustrations of this are seen in certain papules and vesicles which occur upon the palms and soles. These lesions are essentially papules and vesicles, but owing to the unusually thick horny layer which covers them, they are bound down so that they produce no elevation of the surface.

Stains.—A stain is a discoloration of the skin due to abnormal deposit of coloring matter as the result of a pathological process.

Various combinations of these elementary lesions occur. Thus a papule may rise out of the center of a macule, forming a maculo-papule; a vesicle may form upon the apex of a papule, forming a papulo-vesicle. In the same way we have papulo-pustules, scaly papules, ulcerating tubercles, and various other combined lesions.

Lesions of the skin, of course, do not constantly conform to types, and so we see many lesions that are on the border line between one type and another. Thus there may be room for question as to whether a given lesion is a large papule or a tubercle, a large vesicle or a bulla, a pustule or a bulla, but such questions are academic, and the possibility of their occurrence does not diminish in the slightest the value of the typical conceptions. The very vagueness of such an expression as, for example, the size of a split pea, is useful as expressing the fact that we have to do with types which are of approximate but somewhat variable size.

The foregoing definitions of the primary lesions are placed together in order that they may be the more easily compared and contrasted. The lesions, however, require more of a description than can be included in a definition.

Macules.—Macules are probably the commonest of the elementary lesions. The term is usually applied to spots not above the size of a finger nail, but it may with propriety be applied to larger discolorations. Macules may be of any shape, but are usually roundish. Their color is various, according to its source, but is most frequently some shade of red. Macules may be divided into two broad classes: first, those due to deposits of coloring matter in the skin, and second, those due to vascular dilatation. The macule produced by deposit of coloring matter in the skin may have its origin in an abnormal deposit of cutaneous pigment or in a deposit of blood pigment from extravasation of blood. The typical pigmentary macule is the freckle. The pigmented macules vary in color from light to very dark brown, and of course do not disappear under pressure; they are usually permanent lesions. In contrast to the pigmented macule, there occur whitish or nonpigmented macules which are due to the lack of pigment in circumscribed areas. The macules due .to extravasation of blood in the skin are bright red to dark red or black in color at their first appearance, but fade with comparative rapidity, changing from purplish to greenish and then to light brownish yellow. They do not disappear under pressure. These lesions are known by the general term *purpura*. When punctate they are called *petechiæ*; when in streaks, *vibices*; when in larger patches, *ecchymoses*.

The vascular macules are those which are produced by an excess of blood in the cutaneous capillaries. Such lesions may be produced by permanent dilatation of the small blood vessels, as seen in telangiectases and in flat vascular nævi. In these the dilated blood vessels can be recognized as such by their tortuous course. The usual cause of vascular macules is hyperæmia. The hyperæmic or erythematous macule is reddish, usually of roundish outline, with ill-defined borders, and disappears on pressure, because its color is due to blood which is still within the vessels and can therefore be driven out by pressure. These lesions when small (up to the size of a finger nail) and more or less generalized, are called *roseola*, and when of this character are frequently symptomatic of constitutional disturbances, like the exanthemata, typhoid, and other septic conditions, and syphilis. A large hyperæmic area is called *erythema*. A hyperæmic border surrounding an inflammatory lesion is called an *areola*.

Papules.—The essential characteristics of the papule are smallness of size and the absence of free fluid. In size they may vary from pinpoint conical elevations to lesions the size of a small pea or lentil. Most frequently they are rounded in outline, but they may be polygonal, angular, or irregular. They may be acuminate, rounded, flat, or umbilicated Their color is most frequently some shade of red, but varies accordin to the material of which they are composed. They are most frequentl due to inflammation. They may be due to neoplasms, to the accumulation of solid excretory substances, or to other pathological condition The papule of acute inflammation is minute, roundish, or acuminate, an surrounded by a reddish areola. This papule is frequently transforme into a vesicle. The papule seen in various subacute inflammatory proc esses is usually larger, of pinkish color, and is apt to be capped by a scal forming the squamous papule. Papules of other types are: the sharpl conical papule, formed by the accumulation of horn cells around th entrance of a hair or sweat follicle; the round whitish or yellowish papul produced by the accumulation of sebaceous matter underneath the horn epidermis in the condition known as milium; and those formed by th proliferation of epithelium in such conditions as molluscum contagiosum

Vesicles.—The essential characteristics of vesicles are smallness size and nonpurulent free liquid contents. In size they vary from lesion hardly perceptible without a hand glass to lesions the size of a small pe They contain ordinarily serum, which becomes mixed with a few leuc cytes; rarely they are hæmorrhagic. Perhaps the only exceptions to th rule that vesicles have serous or hæmorrhagic contents are those forme by the accumulation of sweat under the upper layers of the epidermi At first vesicles are usually tense with glistening, clear contents. If the persist the walls become somewhat flaccid and their contents becom translucent or milky. They are usually globular or acuminate and occasionally umbilicated. They rarely remain vesicles long; they either rupture, leaving weeping points, or become vesico-pustules or pustules, and occasionally develop into bullæ. If they do not rupture, their contents are absorbed, their bases become cornified, and their epidermal covers exfoliate. When they occur upon surfaces where the horny layer is unusually thick, as upon the palms and soles, the resistance which the horny layer presents above may cause the vesicle to be imbedded and without elevation. In such cases the vesicle appears as a minute collection of clear or slightly turbid fluid beneath the horny layer. In such instances the roof usually persists until the contents are absorbed and ultimately exfoliates. Where vesicles occur closely aggregated, as is usual in acute inflammation of the skin, their rupture produces raw weeping surfaces which may be of wide extent. They are usually unilocular, but may be multilocular.

Pustules.—Pustules[1] differ from vesicles chiefly in that they contain pus. They are always of inflammatory origin and consequently have a red areola. At times there may be pronounced induration of the base. They are yellowish in color except when hæmorrhagic, when they are dark red to black. They may be superficial or deep seated. The superficial pustule is quite analogous in formation to the vesicle; the deep-seated pustule is really a minute abscess in the corium. The superficial pustules rupture readily with discharge of their contents, which dry into yellowish-brown or dark crusts. The deep-seated pustule points upon the surface and upon rupture leaves a small cavity which heals like any other abscess with the formation of scar tissue. The superficial pustule is followed by a certain amount of temporary staining like most other inflammatory lesions, but only in very rare instances by permanent scarring.

Inflammatory papules and vesicles usually itch intensely. Pustules ordinarily itch slightly or not at all, but are usually tender or painful or accompanied by burning. Pustules usually develop from papules or vesicles, but occasionally, as in some cases of dermatitis herpetifor they occur primarily as pustules. The contents of pustules are ser a large number of broken-down leucocytes, and a greater or less n ber of erythrocytes. Of course they are most frequently caused by p forming bacteria, but they may be due to other positive chemota agents.

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[1] It must be said that most authors in their definitions do not limit the size of pustules to approximately the dimensions of vesicles, but define as pustules lesions as large as bullæ with purulent contents. In usage, however, the term pustule is ordinarily applied to the smaller lesions, and the lesions of the size of bullæ are, though they contain pus, still described as bullæ. Thus Crocker defines vesicles as being with "free contents of serous fluid," and blebs as "vesicles which are as large as or larger than a pea," and pustules as "differing from vesicles and blebs only in containing pus." In his description of bullæ, however, he says of their contents that "sometimes there is sero-pus or *pus*," and this statement, although inconsistent with his other definitions, is in accordance with established usage. The lesions above the size of a finger nail containing pus are ordinarily spoken of as bullæ, not as pustules.

Bullæ.—Bullæ differ from vesicles and pustules in that they ar larger size. They may contain serum, sero-pus, pus, or blood. Th maximum size is indefinite. Spontaneous bullæ may be as large a goose egg, and traumatic bullæ from burns may be of almost any s Ordinarily bullæ are at the beginning globular with tense walls translucent contents, but they may be flaccid or turbid from the st In certain conditions they rupture readily, but their walls are usu thick and their contents are likely to be absorbed without rupture. T may occur either upon an indurated or nonindurated base, and th may or may not be an inflammatory areola around them. They are so likely to occur in groups as vesicles and pustules, but when they so occur they are apt to coalesce into large irregular lesions. They develop from other lesions, but they are likely to occur as primary lesi being preceded only by transitory hyperæmia. Ordinarily the sub tive symptoms accompanying bullæ are insignificant—a feeling of sion or slight burning—but in certain conditions the subjective sy toms are much more pronounced and they may itch intensely. mechanism of the development of bullæ is the same as that of vesi They, however, represent a more intense process, either as a result more active local reaction or of a reaction in an individual of low vitality; spontaneous bullæ are, accordingly, suggestive of syste disturbances of considerable gravity.

Tubercles.—The essential idea associated with tubercle in der tology is a solid pathological formation of the skin—not necessarily vated intermediate in size between a papule and a tumor. Tuber may be of the same pathological character as papules, and in color, sh and outline they vary as do papules. The term tubercle, however, is u most frequently to denominate the lesions of the skin which occur in fectious granulomata, and is especially applied to such lesions as t occur in lupus, leprosy, and late syphilis. The tubercles of infect granulomata are situated in the corium or subcutaneous tissue, are s gish in development, and are apt to be persistent. They may ulcer leaving scars, and they frequently undergo involution without ulcerat but with the production of scars. The term tubercle is most usefu an appellation for these neoplastic lesions, and it has been propose limit its use to them. Granulomatous tubercles may be quite imbed in the skin and without elevation, as frequently seen in discrete les of lupus. Ordinarily they are elevated, and where they are numerous they tend to coalesce and form plateaux of elevated skin. An objection to the use of the term tubercle is that it is apt to be assumed as indicating an association with tuberculosis or the tubercle bacillus; the term nodule has been suggested by Crocker, and may be used as a substitute for it.

Gummata.—The gumma is a lesion of the same pathological character as the granulomatous tubercle but of larger size. The term gumma is applied especially to large subcutaneous lesions of syphilis, and also to similar lesions of tuberculosis. Gummata are situated deep in or beneath the corium, and do not at first involve the superficial layers of the skin. They may develop rapidly or slowly. They show as obtuse, rounded elevations of the skin, either of normal color or some shade of red. At first they are hard, then tend to become boggy in the center, and finally may open with deep central ulceration. Or they may terminate by absorption without ulceration.

Tumors.—The term tumor may be technically applied in dermatology to any solid lesion above the size of a hazelnut. Its most frequent application, and the applications to which usage is tending to confine it, is to new growths of the skin regardless of size.

Wheals.—Wheals are the characteristic lesions of urticaria, and their minute consideration will be taken up under that subject.

Scales.—Scales are exfoliations of the epidermis. They may vary in size from fine, branny scales to great sheets of horny epidermis. In thickness they vary up to masses which may be half an inch thick, as seen, for example, in exaggerated psoriasis. Their character, of course, varies according to their cause. They are dry and brittle and whitish unless mixed with exudation, when they are likely to be soft and grayish and of a rather greasy feel. Their quantity may be limited to a few minute desquamating points of epidermis upon a papule, or they may form over the entire body to the amount of a gallon or more in twenty-four hours. They are the result of inflammation in the skin, usually of inflammation which stops short of the formation of vesicles or of the collection of serum in free cavities in the epidermis. Hyperæmias, except the most transitory, are followed by branny desquamation, the best examples of which are seen in the desquamation following the exanthemata. In all of the chronic inflammatory lesions of the skin scales occur in greater or less abundance, and in some dermatoses, such as psoriasis, they are the chief characteristic of the disease.

Crusts.—Crusts are due to dried accumulations of all sorts upon the skin. They are most frequently produced by the drying of pus, serum, blood, and epithelium in varying admixtures. They may be due to accumulations of fat with epithelium and dirt, as in seborrheic eczema, or

to accumulations of microörganisms, as in favus. Occasi
of distinct diagnostic importance, as in favus and syp
have been studied with minute attention. But, as Brocq
ally the most important thing pertaining to crusts in
necessity of removing them in order to examine the underl

Excoriations.—Excoriations are abrasions of the skin,
deeper than the mucous layer, but frequently involving
the upper surface of the corium. They are usually the res
ing. They ordinarily heal without the production of scars,
that the connective-tissue structure of the skin is not i
involved only very superficially. Their size, shape, and char
upon the mode of their production. Excoriations due to sc
of distinct diagnostic importance. In the first place, they
an intense degree of itching; if punctate, they indicate that
papules have been scratched off; if in short lines, usually tha
been a flat, elevated lesion, like a wheal, of approximately the
of the scratch mark; if they are long lines—half an inch or
length—and deep, they indicate a very intense type of itching,
seen, for example, from the invasion of parasites like body lice.

Rhagades.—Fissures in the skin occur upon surfaces which
posed to motion and which, from an inflammatory or some other
logical process, have lost their normal elasticity. They are conseq
seen especially about the mouth and anus, the palms and soles, an
extensor surfaces of joints. They occur in the lines of cleavage o
skin for the very simple reason that they are produced by the same m
ments that determine the direction of these lines of cleavage. In cer
circumstances, as in the fissures about the mouth in congenital syph
they are of great diagnostic significance. They usually heal with
ulceration, but if they are accompanied by considerable loss of subst
they may be followed by linear scars. They usually cause more or
pain on motion.

Ulcers.—Ulcers are most frequently the result of infection, with
nary pus organisms, of superficial wounds of the skin or of the folli
They are particularly prone to occur as the result of the infectio
abrasions upon surfaces where the skin is permanently congested,
varicosed legs. Aside from infection with the common pus organi
they occur most frequently as a result of syphilis, carcinoma, or t
culosis. The character of ulcers is of extreme diagnostic import
For example, the ulcers of syphilis, of carcinoma of the skin, of l
present characteristics which are almost or quite pathognomonic.
points about them which are especially to be observed are the char
of their borders and floors, their shape, depth, location, course,
excretion.

Scars.—Scars are invariably the result of loss of substance of the corium. This most frequently is produced by ulceration, but it may occur as a result of a destructive pathological process which disappears by absorption without ulceration. In the formation of scars the papillary structure of the corium, the hair follicles, and the glands disappear, and there is usually left a layer of connective tissue separated by a plain surface from a thin overlying epidermis. The extent of the scar tissue formation that follows different ulcers of the skin varies considerably, with the result that we have thin, white, atrophic scars ("cigarette-paper scars"), thick, bandlike scars, or hypertrophic scars which may amount to tumors, as in keloids. In the formation of scars there is always more or less contraction from the size of the original lesion. This is particularly true of thick scars, so that where scars are extensive they may cause great deformity and interfere materially with the mobility of the parts. The character of scars is of almost as great diagnostic significance as that of ulcers, and careful attention is to be given to their location, shape, texture, color, and other features.

Stains.—Stains are most frequently the result of the deposit of blood pigment in the skin in the process of inflammation. They may be produced by an increased formation of pigment as the result of a persistent superficial dermatitis, as in sunburn, or by the deposit of extraneous insoluble pigments in the corium, as in powder stains or tattoo marks. They occur with particular frequency as a result of the deposit of blood pigment after chronic inflammatory lesions, as syphilides, lichen planus, psoriasis.

ERUPTIONS

Composition and Configuration of Eruptions.—The combination of lesions of the skin which occurs in any given case constitutes an eruption. An eruption may consist of lesions of one type, when it is called uniform; but it is much more frequently composed of lesions of different types, when it is called multiform or polymorphous. The lesions of an eruption may be discrete or they may be collected in circumscribed areas, forming patches.

There are many terms used technically to describe the configuration of eruptions. We speak of an eruption as punctate when it occurs in small discrete points; as guttate if the lesions are of the size of drops; as nummular if the size of a coin; as lenticular when the size of a split pea or bean; as marginate when having sharply defined borders; as circinate or annular when rounded or in ring form; as gyrate when occurring in winding forms; as serpiginous when creeping or spreading at the border; as corymbose when several lesions are grouped like satellites around a central lesion; as herpetiform when occurring in aggregated groups, like herpes.

When lesions combine into patches they may assume an appearance which is superficially quite different from that of their component elementary lesions. In a general way, the patches show in an exaggerated degree the characteristics of the elementary lesions. For example, a patch formed from the coalescence of scaly papules will likely be thicker, more indurated, of darker color, and with a greater abundance of scales than the individual lesions. A patch formed from the coalescence of tubercles, as for example, those of lupus, will likely be thicker and show more scaling and hypertrophy of the epidermis than the individual lesions. In a suppurating patch formed from the coalescence of pustules, the base is likely to be more indurated and the pus crusts thicker than in the discrete pustules. It may be difficult to determine the mode of evolution of a patch, and information concerning this can usually be gained from an examination of the discrete lesions, which are likely to be found like satellites around the border of any patch. These satellite lesions are, as a rule, the elementary lesions of the process.

From the grouping of lesions in patches deductions can frequently be drawn as to their mode of development. For example, a thickened patch with sharply defined convoluted borders, around which there are discrete papules or tubercles, usually means that the lesion has developed from the confluence of a group of papules or tubercles. A round or annular patch with sharply defined borders, and with a center which is fading, indicates that the lesion has been formed from a smaller central lesion, which has spread peripherally while it was undergoing involution at the center. Such peripheral extension and central involution of a pathological process is a very characteristic and not uncommon occurrence in skin diseases, and gives rise to striking clinical pictures. If several such spreading rings occur near together their borders in time usually meet. When this occurs it is the rule that at the points of contact the process subsides, and so larger lesions are formed whose borders are made up of segments of circles. It is always in this way that gyrate figures are formed. Serpiginous ulceration in the same way produces ulcers with scalloped borders, and eruptions like psoriasis and ringworm, which tend to spread peripherally, frequently produce fantastic gyrate figures of large size, in which the borders show the active process, while large areas of inclosed skin may be quite, or almost, of normal appearance. Occasionally an eruption will occur in a spreading ring, and after it has progressed to a certain size a second similar lesion will develop in the center; this will also spread peripherally, forming a second circle around the same center; and perhaps a third ring or a central lesion will develop within this. In this way *iris* figures develop.

Secondary Changes in Eruptions: Secondary Infection; Lichenification.—The essential character of an eruption may be obscured by accidental complications; the most common of these are secondary infection and traumatism as a result of scratching. Secondary infection almost invariably occurs, unless guarded against, in all eruptions in which the epidermis is lost. In ulcers it may, by superimposing an acute inflammatory process and causing the abundant formation of pus, greatly alter the original appearance. In eruptions where the surface is simply denuded of epidermis, the usual manifestation of secondary infection is the occurrence of pus and the resulting formation of pus crusts. It is very frequent in such cases for boils to result; occasionally ulcers and cellulitis intervene; and very rarely infection with the specific organisms of syphilis or tuberculosis or of some of the rarer infectious diseases occurs. In these latter instances, of course, a new disease is superimposed upon the original pathological process. The effects of infection with the ordinary pus organisms must be constantly looked out for and gotten rid of. Frequently it happens that when this is done we find that what we thought was a dermatosis of another character has disappeared; or, if this does not happen, the true character of the essential process becomes plain.

In itching dermatoses the results of scratching may alter markedly the appearance of the eruption. The scratching excites an inflammatory process, which, if persistent and not too acute, results in a thickened induration of the involved skin. This subacute inflammatory induration of the skin has been called by Jamieson *leathery induration*, and by Brocq *lichenification*. Jamieson's term is more descriptive of the condition and is not confusing, but Brocq's term bids fair to become es-

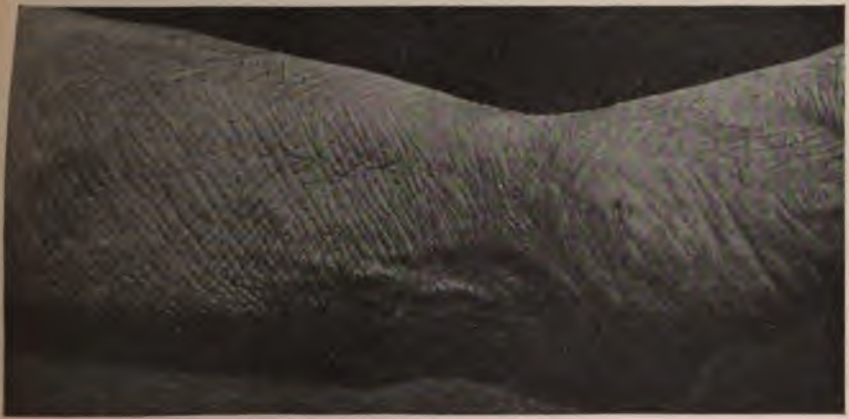

FIG. 28.—LICHENIFICATION OF SKIN OF FOREARM. (Brocq.)

tablished in dermatology because of the minuteness and accuracy with which he has described the condition. The essential feature of lichenification is persistent subacute dermatitis. It occurs most frequently as a result of scratching in itching dermatoses. It may be produced from any external irritation, like the friction of clothing or the contact of irritating secretions. Under the persistence of the inflammatory process there is an increased deposit of pigment in the skin; the partially organized inflammatory exudate makes the skin thick and leathery; the normal surface markings are consequently exaggerated and become converted into linear furrows; and the minute areas of skin between these linear furrows appear as flat, angular papules, simulating the angular glistening lesions of lichen planus. The confusing feature about these patches is the appearance they have of being made up of closely aggregated angular papules. This appearance is due to the exaggeration of the normal markings. The angular pseudo-papules are not the elementary lesions of the process, but are only the result of the exaggeration of the normal markings of a skin thickened by a diffuse inflammatory process. The picture may be seen in any chronic dermatitis. It is not specific, and is not a manifestation of the definite disease lichen planus.

The appearance of an eruption may be altered by peculiarities of the skin of the individual. The most striking illustration of this fact is found in the masking of the appearance of dermatoses occurring in dark races. In such cases the abundance of pigment quite obscures the ordinary tints of lesions as they occur in individuals like Caucasians with translucent epidermis. This is so completely true in full-blooded negroes that the important factor of color is entirely excluded. Certain peculiarities in individuals occasionally present complications in the development of eruptions. Perhaps the most common of such complications are unusual development or activity of the sweat or the sebaceous glands, or the opposite of these conditions. Occasionally we see a complication arising from the development of an eruption upon an area which is already the seat of a pathological process. One of the commonest illustrations of this is the alteration in dermatoses which develop upon extremities which are affected by passive congestion. In such cases the lesions are darker than normal, are indolent, are more ready to ulcerate, and are apt to be followed by persistent pigmentation. Another alteration in the appearance of lesions is seen where dermatoses develop in cachectic individuals, in whom, from the relative diminution in the quantity of blood, the lesions are apt to be less florid than normal.

We occasionally have one definite dermatosis superimposed upon another. The commonest illustration of this is the development of secondary infection upon a weeping or ulcerating dermatosis, to which reference has just been made. Another example, but one of greater rarity, is the development of epithelioma in a scar of lupus or a patch of psoriasis. There is occasionally seen a combination of very practical importance between the eruption of syphilis and seborrhœa or seborrhœic dermatitis. When combinations of dermatoses occur one usually finds a solution of the difficulty of diagnosis in the fact that the combination does not extend to all lesions, and at different points will be found uncomplicated lesions of one process or of the other.

Color.—Color is an essential factor in the picture of all eruptions, and in many eruptions it is more or less characteristic. The predominating tint is, of course, some shade of red. This varies from the light rose tint of an evanescent erythema to the blood-red color of an inflamed surface denuded of epidermis, or the dark violaceous color of cyanotic skin. Certain shades are distinctive of certain conditions, as the raw-ham color of secondary syphilides, the apple-jelly color of lupus tubercles, and the violaceous tint of patches of lichen planus. In extravasation of blood into the skin dark purple to black is seen. Hyperkeratoses produce a brown color of the skin which may vary from a light, dirty, brownish superficial discoloration to black horny masses. The growth of fungi upon the surface of the skin, if unaccompanied by apparent inflammatory reaction, is likely to produce brownish discoloration similar to that of moderate keratosis, and in exaggerated instances this accumulation may even be black. In certain conditions peculiar coloration of lesions occurs. Thus, in the disease xanthoma the lesions are chamois yellow; in favus the peculiar crusts that form are of a sulphur yellow; as the result of pigment-forming bacteria on the skin, such shades as blue and green are occasionally seen. But such colors are anomalies, and the colors of most dermatoses are some shade of brown or red or combinations of the two.

Distribution and Localization of Eruptions.—The limits of distribution of eruptions[1] vary from a single elementary lesion to eruptions which involve the entire integument. The various general forms, however, in which eruptions occur are relatively few. The lesions may be collected in a patch upon one part; they may be irregularly scattered over the body, when they are spoken of as disseminate; they may be generalized, that is, distributed over all parts of the body, but not involving the entire integument; they may be symmetrically or unsymmetrically distributed as regards the two sides of the body; and in certain rare dermatoses they may involve the entire area of the integument, when the eruption is called universal. Speaking broadly, eruptions which exist only at one point are likely to be due to local causes, and eruptions which are generalized or symmetrical are likely to be due to systemic causes. Symmetry of eruptions especially is supposed to indicate systemic causes; and this is usually true, as illustrated in the eruptions of the exanthemata, in those due to toxæmias, and in those due to central nervous disturbances. But the rule is by no means universal. The fact that an eruption is generalized only indicates that it is due to causes which are exerting their effect over the general surface of the body. These are most likely to be internal, but they may be widely distributed external causes, as parasites in clothing or in the skin. In the same way symmetrical eruptions may be due to local causes which from their nature affect corresponding parts of the body; thus causes arising in wind and weather, or in occupations, are likely to affect equally the exposed parts on both sides.

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[1] Sabouraud, "Topographical Dermatology," Rebman, London and New York, 1906.

The influences which determine the distribution and localization of eruptions are many. These are likely to be complex in any case, but it is often not difficult to discover the predominating influence which determines the particular distribution of a given eruption. These factors may lie in adventitious circumstances, like especial exposure of certain parts, the effects of clothing, of occupation, or of habits. Or they may lie in intrinsic characteristics of the skin of certain parts from anatomical or functional peculiarities.

The hands and face, from their lack of covering, are manifestly particularly likely to be the site of eruptions arising from the effect of light and heat and contact with external irritants of all sorts; and the effect of these factors in distribution of eruptions on these parts is constantly to be seen. The parts of the body that are covered by clothing, on the other hand, are the characteristic sites of distribution of other eruptions which are favored by this fact. We see illustrations of these in eruptions produced by the friction of clothing, by the harboring in the clothing of parasites, or by the favoring influence of warmth and moisture and the accumulation of irritating secretions which the clothing affords. In addition there are many eruptions which appear upon the clothed parts and do not appear upon the uncovered parts, in which the factor of protection by clothing seems to be of importance, although the connection may not be apparent.

The functional and anatomical peculiarities of different parts of the body are easily recognized factors in the distribution of many eruptions. Perhaps the hands and feet illustrate best this fact; they are especially exposed from the uses to which they are adapted to the effects of external irritants; they are anatomically peculiar in that they are covered with unusually thick epidermis; and they represent distal vascular areas. It is not surprising, therefore, that they are special sites of distribution of many eruptions depending upon these various factors

The orifices of the body, partially from the delicate texture of the skin surrounding them and also from their peculiar exposure to mechanical and chemical irritants, are especial sites of distribution of eruptions. Those parts of the body which are abundantly supplied with glands, or which from their contour favor the accumulation of the secretions of the skin upon them, like the axillæ, the fold of skin below the breasts, and the genito-crural fold, are the especial sites of distribution of certain dermatoses. Again, the abundance or absence of hair is a determining factor in the distribution of some eruptions. The peculiar texture of the skin of certain parts is another factor. The skin of the flexures of the body and at the muco-cutaneous junctures is delicate and thin. It is consequently less able to resist insult than skin of thicker texture, and these sites are accordingly points of predilection in the distribution of certain dermatoses. The vascular peculiarities of different parts are probably factors of importance in the distribution of many dermatoses, although the fact may not have been worked out. Upon certain parts of the body, however, this fact is clearer. The area of the body where the vasomotor mechanism is most sensitive is the so-called flush area of the face. The points of greatest intensity of this area are the nose and the center of the cheeks; from these points the area spreads with diminishing sensitiveness over the rest of the face. In the distribution of several eruptions the influence of this peculiar vasomotor area is apparent; in many others it is intimated. Crocker suggests as other important vascular areas: the entire head and neck; the forearm, back of the hand, and lower two thirds of the upper arm on the extensor surface; and the so-called bathing-drawers area, that is, the lower third of the trunk and the upper parts of the thighs. But while these are common areas of distribution of dermatoses, the importance of the particular vascular factor is not established. The influence of distal areas of circulation in the distribution of eruptions is seen in the predilection of the hands and feet, already referred to, and of the ears and nose. This influence is particularly seen in the distribution of eruptions on the legs, where the circulation has to overcome not only the obstacle of distance, but gravity as well.

What has been said of vascular areas applies in large part to nervous areas, because of the control which the nervous mechanism exerts over the blood supply; but independent of this we see eruptions whose distribution is plainly determined by nervous control. The most striking illustration of this is seen in herpes zoster. It is suspected but not capable of demonstration in many lesions which follow the distribution of cutaneous nerves.

The influence of the lines of cleavage of the skin in determining the distribution of eruptions has been minutely investigated, but the influence of this factor, as distinguished from the influence of other more easily recognized factors which come into play upon the same parts, has not been very clearly made out. The influence of the lines of cleavage of the skin upon the configuration and grouping of patches of eruptions is more capable of demonstration and is apparent in many lesions.

SUBJECTIVE SYMPTOMS

Disturbances of Temperature.—The temperature of the skin of circumscribed areas may be reduced as the result of passive congestion. This occurs especially from poor peripheral circulation, and is usually seen in the hands and feet and occasionally in the ears and nose; the parts are cold and clammy to touch, and feel cold to the patient. Increased temperature of circumscribed areas may result from active hyperæmia. The temperature in such conditions is actually above that of the unaffected areas, but, of course, not above that of the blood. Accompanying this elevation of the temperature of the skin, there is to the patient a sensation of heat which in rare instances is of extreme intensity.

Disturbances of Sensation.—The normal sensibility of the skin may be increased (hyperæsthesia), decreased (hypæsthesia), or abolished (anæsthesia). These are all among the rarer subjective phenomena in the skin, and are associated with functional or organic nervous disturbances. Cutaneous hyperæsthesia is characterized by exaggeration of the normal sensibility of the skin, which may attain the point where touching or rubbing the skin causes severe pain. Cutaneous anæsthesia may be complete, with loss of both touch and temperature sense, or it may involve one without affecting the other. Cutaneous hypæsthesia may likewise be complete, involving both touch and temperature sense, or it may be partial, involving one or the other. Both anæsthesia and hypæsthesia are usually confined to more or less sharply limited areas. It occasionally occurs that anæsthesia, or hypæsthesia, and hyperæsthesia are successively manifested in the same area.

Painful Sensations.—Painful phenomena in the skin may be divided, following Brocq, into true painful sensation and pruritus. It is not always easy to dissociate in a given case these two classes of sensations, but in their extremes of manifestation they are quite distinct. Painful sensations in the skin include the sensations of warmth, burning, smarting, tension, constriction, and typical pain. The sensations of warmth, burning, smarting, and prickling are usually associated with acute inflammatory processes in the skin in which there is tension. The sensation that we ordinarily understand by the word pain is not of common occurrence in the skin, but does occur both as sharp lancinating pain and dull aching pain.

Pruritus.—The characteristic disturbance of sensation in the skin is itching. Itching may be regarded as the analogue in the skin of painful sensations in other structures. It is a pathological sensation distinctly associated with the sense of touch. It is a unique sensation which cannot be described in other terms, and a characteristic of it is that it provokes a desire to scratch. The relief of itching which comes from scratching is probably explained by the fact that in the act there is a substitution of one sensation for another. When itching is not intense the substitution of a moderate sensation of pressure or friction is sufficient to allay it; in its most intense manifestations, relief is only obtained by the production of what would ordinarily be painful excoriations. Scratching and itching are thus closely correlated; and the intensity of itching may be accurately judged by the extent of the evidences of scratching which the skin manifests. In its most exaggerated form itching may amount to almost unexampled torture. Itching is commonly a manifestation of a vasomotor or inflammatory process in the skin. It may occur without any appreciable pathological process in the skin at any point, and is of very common occurrence in areas of normal skin as the result of reflex irritation from itching lesions existing on other parts of the body.

VI.—DIAGNOSIS

Means of Examination.—The objective symptoms of cutaneous diseases are chiefly those which are appreciable to sight. The sense of touch, also, though much less frequently, gives valuable information. In a few rare conditions, like favus and pemphigus foliaceous, we obtain information of diagnostic importance through the sense of smell.

There are few instruments for the dermatologist to use in making his examinations of the condition of the skin. The hand lens, magnifying from two to three diameters, is of great assistance in the examination of individual lesions, and its habitual use should be cultivated. The typical characteristics of many minute lesions can be well made out only with the aid of the magnifying lens. To determine the character of infiltrations in the skin it is often desirable to examine lesions from which the blood has been expressed. For this purpose a smooth piece of glass is necessary, through which the lesion can be examined while pressure is being exerted. Special instruments for this purpose are made—Hess's pleximeter, for example—but nothing serves the purpose better than a plate-glass spatula, and for occasional use a hand lens or an ordinary piece of glass is sufficient. To obtain tissue for microscopical examination a cutaneous punch is sometimes used, a sharp round punch about one fourth to three eighths inches in diameter. This has the advantage of giving a solid plug entirely through the skin which is convenient for mounting and cutting. But its use is more painful than the excision of a wedge-shaped piece, and is followed by more scarring. In taking tissue for examination, it should not be pinched up with forceps and snipped off with scissors, but an incision should be made with a small blade along the lines of cleavage around the lesion which is to be excised, and after this is done the piece should be raised and cut off evenly. This, of course, can be made painless either by the injection of a few drops of one-per-cent solution of cocaine or eucaine, or by freezing with ethyl chloride.

The use of the microscope both for the examination of tissues and for the study of microörganisms must, of course, be constantly invoked.

Examination of Patients.—The ideal conditions for the examination of a cutaneous eruption require good diffused daylight and a room the temperature of which is not cool enough to chill the patient. Faint shades of color, especially of yellow and brown, may not be appreciable in artificial light, and the entire appearance of pronounced eruptions is likely to be considerably changed. So true is this that it is not safe to depend upon the examination of doubtful eruptions under artificial light. If an examination is to be complete, it is desirable that the room should not be cool enough to chill the patient, for the reason that sudden chilling of the surface on exposure not only causes a temporary anæmia of the skin, from the contraction of its involuntary muscle fibers, which is sufficient to obscure faint erythemas, but causes an exaggeration of the normal mottling of the skin—the so-called marmor cutis—which may alter the appearance of erythematous lesions or even suggest an eruption which does not exist.

It is a good practice, but one which has to be exercised with tact, to examine habitually the entire eruption. The partial examination of an eruption is often confusing, and it frequently happens that the character of an eruption which in a given patch is obscure becomes plain when other lesions are seen. A more satisfactory picture of an eruption is gotten by complete exposure of the body at one time, but if this cannot be obtained a fairly satisfactory picture can be made up by the examination of different areas successively. In examining eruptions piecemeal care should be taken that parts of them are not overlooked. Unless patients are intelligent, they are apt to deny obstinately the existence of areas of eruptions which are not giving them annoyance or which, if they think about them at all, they suppose are not important.

Upon the first examination of a patient a general mental inventory should be taken. To the practiced eye this quickly gives information;

to the less experienced the same information may be obtained if this inventory is made slowly but thoroughly. It is the constant experience of every expert that most failures in diagnosis in dermatology, as well as in other departments of medicine, are made, not because facts are difficult to elicit, but because they are overlooked. The items to which attention is given in the general inventory of the patient are age, sex, social conditions, general aspect, physical condition, complexion, temperament, and other such features. After this we turn to the history of the patient and the history of the dermatosis. Under the first are to be considered the family history of the patient, his previous history as regards health and disease, habits and occupation, the present condition of his health; under the second, the duration of the eruption, its course, its distribution and extent. And after this we come to a minute consideration of the lesions. It is often true that the essential characteristics of an eruption so obtrude themselves that in a given case no such systematic examination is made, but this does not lessen the value of the habit of systematically considering all of the factors which are essential to a complete examination.

The rôles which these various factors play in the symptomatology and etiology of dermatoses have already been considered under the subjects of general symptomatology and general etiology. Nothing can be said upon the subject of general diagnosis in dermatology, which is not a question either of symptomatology or of etiology. The capacity to make a diagnosis is, after all is said, a question of knowledge of symptomatology and etiology, and the chief point to be made here is to emphasize system and thoroughness of examination in order that the knowledge which the practitioner possesses may not be wasted.

VII.—TREATMENT

The treatment of skin diseases is both external and internal; a discussion as to the relative importance of these is as profitless as a discussion of the relative importance of the arms and legs or of any other essential things. At one time both external and internal treatment may be important, at another time the one or the other. In diseases like syphilis, in which the cutaneous manifestations are but a part of the general disease, internal treatment is essential; in other diseases which are dependent solely upon external causes, external treatment may be all that is needed; between these two extremes situations arise in which the two methods of treatment present all degrees of relative importance. External treatment, however, is more peculiarly dermatological and is of more frequent application, for even in diseases of the skin due solely to internal causes there are few instances in which external measures are not needed.

The success of both internal and external methods of treatment rests on the one hand, upon a proper appreciation of symptomatology, etiology, and pathology; and, on the other, upon the intelligent application of remedial measures. In dermatology, as in medicine generally, we have few specifics. In certain diseases it has been found empirically, and, in a very few brilliant instances by deduction from the physiological action of drugs, that certain remedies have beneficial effects which may be said to approximate specific curative properties. But, if we except the iodides and mercury in syphilis, the list of internal specific remedies of established value is so small as to add greater emphasis to the statement that successful treatment depends upon the rational application of therapeutic measures.

Both internal and external methods of treatment are directed as far as possible to the removal of causes. Diseases in which the essential causes are known and removable offer the ideal opportunities for treatment. To take a gross illustration: diseases which are due to known animal parasites are readily cured by measures which destroy the parasites. But the usual situation which confronts us in therapeutics is not nearly so simple, and if successful treatment were dependent upon a complete knowledge of pathogenesis, our therapeutic successes would be very much more limited than they are. In some diseases we can only approximately determine the causative factors, and they may then be very complex; in many others they are entirely unknown. In such situations as these treatment has to be directed to meeting by rational therapeutic measures the indications which are derived from the symptomatology and pathological histology. Fortunately in the majority of such cases intelligent treatment so directed will be successful; for when by treatment we succeed in the relief of symptoms the skin is usually able to restore itself to normal.

The number of agents which are recommended for various diseases of the skin is almost innumerable, but the number of remedies that it is necessary to use in order to meet all of the pathological conditions that we are capable of combating is not large. The actions of many therapeutic agents are similar or overlap each other, and of the agents of similar properties experience has shown that a few are of the most practical value. These comparatively few agents and methods of established value form our main reliance in meeting almost all of the multifarious conditions of cutaneous diseases which confront us. Each practitioner finds by experience that the means of treatment which he uses narrow themselves in time down to relatively few with which he has familiarity, and with which he works with intelligence and assurance. A firm grasp of the well-established means of general and dermatological therapeutics, rather than a hazy knowledge of the innumerable agents which are recommended for the cure of this or that disease, is the essential of success in the treatment of cutaneous diseases. There are few things more futile than the aimless use of remedies with whose properties one is not familiar in an endeavor to find the specific remedy for a particular disease or case. As Sir William Broadbent, I believe, put it, new remedies are invented every day, chiefly for the benefit of those who do not understand the use of old ones.

The aim of treatment, of course, is to cure diseases, and not many cases arise in diseases of the skin in which we need fear, on account of our patient's safety, to attain this end as quickly as possible. The question, however, sometimes does arise whether there is not danger of "driving in" an eruption. The possibility of such danger is frequently the refuge of defeated incompetence, but the question may be asked in all honesty. It is the almost universal opinion of experts that there is no danger of getting rid too quickly of any eruption. Such observers as Hebra, Unna, Duhring, and Crocker, believe that there is no such danger, and it is hard to find any theoretical grounds for fearing it.

INTERNAL TREATMENT

Under etiology we referred to the various constitutional conditions which influence or produce diseases of the skin. The internal treatment of skin diseases is for the most part the treatment of these underlying conditions, and is carried out, it need hardly be said, in the same way as when they are not associated with diseases of the skin. In skin diseases we are called upon most frequently to correct disturbances of digestion and of metabolism and the intoxications resulting therefrom. Disturbances of the nervous system also demand much attention. Disturbances of digestion and of metabolism are so frequently factors in the etiology of the most common skin diseases that their treatment forms, perhaps, the largest part of the internal treatment of diseases of the skin. The treatment of the various conditions is a part of general medicine.

There are, however, certain measures which are entitled to special consideration because of the frequency of their use or because of the special application which is made of them in dermatology. They may be broadly divided into two classes:

(1) Remedies which are used for their indirect effect in correcting constitutional defects, like tonics, aperients, intestinal antiseptics, hæmatopoietics, and general hygienic measures, as rest, diet, massage.

(2) Remedies which are used specifically for their effects on the

skin or on pathological lesions in the skin, like mercury, the iodides, arsenic, antimony, sulphur, phosphorus, jaborandi, tar, creosote, carbolic acid, guaiacol, quinine, resorcin.

The specific usefulness of a few of these agents, like the iodides, mercury, and arsenic, is undoubted; that of most of the rest is at least problematical.

Hygiene; Climate; Rest; Diet.—The indications for special attention to these general measures are for the most part the same in diseases of the skin as in other diseases. In certain diseases special emphasis upon one or the other of them is at times very highly important. More than ordinary attention to the principles of right living is called for in various diseases which are persistent or tend to recur, and which are excited by almost any departure from the normal balance of health. The cases are many which are excited or exaggerated by carelessness in eating, by lack of attention to regularity in movements of the bowels, by physical or nervous overexertion, by lack of sleep, or by other hygienic peccadilloes. The indications for special attention to these matters is only found by careful examination into the methods of living of patients.

Those diseases which are exaggerated in summer or in winter are benefited by removal to a suitable climate—a measure, however, which is not available for most patients. The ordeal of a long, cold winter, particularly if it is characterized by cold winds and much cloudy and rainy weather such as prevail around the Great Lakes of North America, is exceedingly trying to certain cases of diseases of the skin, and at times removal to a softer climate becomes imperative if relief is to be obtained. The benefit of such changes of climate is seen in many cases of chronic inflammatory diseases of the skin, like eczemas of the face, and in other conditions in which peculiar susceptibility to cold seems to be a characteristic feature, like pityriasis rubra. In cases of this sort removal to a more suitable climate often gives complete relief. The necessity for avoidance of a summer climate in diseases of the skin is not so pressing. There are some dermatoses which are worse in summer, but they are ordinarily not of the serious or distressing character which renders relief imperative.

The usual indications for rest obtain in diseases of the skin, but the benefits to be derived from complete rest are especially marked in some conditions. In extensive acute eczemas, for example, and in other extensive inflammatory disturbances the patients do best when they are put to bed. While dragging themselves around, even if they are not undertaking to perform their usual duties, the disease may resist every effort at relief, but yield rapidly when the additional measure of rest in bed is taken advantage of.

The regulation of the diet[1] in diseases of the skin is a particularly important matter, because of the frequency with which dermatoses are connected with disturbances of digestion. The indications to be met are the usual indications in these disturbances. The attempt to regulate diet with specific reference to the effect upon the skin, which is sometimes attempted, is of doubtful utility. For example, there is little reason to believe that the avoidance of fats which is sometimes advised in acne is of any advantage unless fats are contraindicated by positive digestive disturbances.

Alcohol.—The question of the use of alcohol calls for careful consideration in diseases of the skin, for alcohol has a direct effect upon the skin; it causes flushing of the skin, and where there is a tendency to itching it is very apt to excite it. The use of alcohol, therefore, is generally contraindicated in acute hyperæmic conditions of the skin and in itching dermatoses. It is especially liable to be harmful in inflammatory diseases of the face, whether acute or chronic, for the flushing effect of alcohol is most marked upon the face. Alcohol, however, is not to be arbitrarily denied in all dermatoses. In patients who are reduced in health it may serve a useful purpose in whipping up a poor appetite. The moderate use of beer and malt liquors is at times a valuable tonic in anæmic patients with poor digestions, Beer also sometimes serves a useful purpose, when drunk in the evening before retiring, by inducing sleep in nervous patients. The practitioner is frequently confronted with the question of cutting off alcohol from patients who use it habitually. There are certain dermatoses which occur in such individuals entirely independent of the use of alcohol, and in these cases its sudden withdrawal is apt to do harm, just as is the sudden changing of any other habit. Alcohol is best taken at the beginning of or with meals, either in the form of malt liquors, light dry wines, or spirits well diluted.

Mineral Waters.—The natural mineral waters have no especial indications in diseases of the skin. Aperient and diuretic waters are useful in the treatment of the intestinal and metabolic disturbances which underlie diseases of the skin, but their usefulness depends entirely upon their general effects and not upon any specific action which they exert upon the skin. The ferruginous waters offer no advantage for the administration of iron. The waters containing arsenic and those containing bromine and iodine, which are found in a few European springs, do not, in the opinion of disinterested observers who have watched their use, show any remarkable virtues. They are, to use the phrase of Crocker, especially "adapted for weak digestions and long purses."

The claims of remarkable specific virtues in various skin diseases which are made for natural mineral waters, particularly as they are used at their springs, are greatly exaggerated. The benefits that are derived are chiefly those that come from repeated purgations, regulated diet, changed and often agreeable surroundings, rest, and regularity of living. Excepting the aperient and diuretic waters it is probable that the greatest benefit is due to an ingestion of an increased quantity of water. In my opinion no weight is to be attached to the idea seriously suggested that the natural mineral waters possess some mysterious potency from the fact that they are made in " Nature's laboratory," or that they possess any greater remedial virtues than waters of the same composition of artificial mixture.

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[1] J. C. White, *Trans. Amer. Derm. Assoc.*, 1887.—Brocq, *Jour. Cutan. Dis.*, Mar., 1896.—Jamieson and W. G. Smith, *ibid.*—*Discussions Trans. Amer. Derm. Assoc.*, 1906.

Analgesics, Antipruritics and Sedatives.—For the relief of pain in skin diseases the ordinary anodynes are used. Where relief is imperative morphine, as always, is the reliance, and fortunately in those dermatoses where morphine is indicated, as in painful zosters and epitheliomata, there are no especial contraindications; the only objections to its use are those which obtain at all times. When we do not want to go to the extent of using morphine, we depend upon codein, phenacetin, aspirin, antipyrin, and similar analgesics. The necessity, however, for the relief of pain is not a frequent condition in diseases of the skin. The subjective sensation, which is of constant occurrence in skin diseases and for whose relief the call is incessant, is itching, and unfortunately we have no internal remedies that we can use for the relief of itching with anything like the certainty with which anodynes can be used for the relief of pain. Morphine, even when the situation is so insistent as to put away the general objections to its use, does not fill the demand, because it produces in many instances itching and, further, because short of narcosis it does not satisfactorily control the sensation. For relief of the nervousness which accompanies itching dermatoses, and to a certain extent for the relief of the itching itself, use is made of the usual sedatives—the bromides, chloral, antipyrin, phenacetin, sulphonal, belladonna, and hyoscyamus. The effects of these are far from satisfactory. The use of sedatives is regarded by many dermatologists with disfavor, and if they are prescribed freely, they are apt to be abused. They are, however, of distinct service at times in itching dermatoses, when the patients are highly irritable and nervous. In such cases the bromides, chloral, sulphonal, and antipyrin, when carefully used, may perform a useful function. I believe also that the action of valerian and asafœtida in relieving nervousness may sometimes be made use of to good advantage. Antipyrin among the coal-tar anodynes has been particularly recommended for the relief of itching by Blaschko, and is, perhaps, the best of these preparations for this purpose.

Mercury.—The preparations of mercury, of course, have their most important function in dermatology in their specific use in the treatment of syphilis. The mercurial cathartics are also in constant requisition in cleaning out the intestinal tract in the frequent dermatoses which are associated with gastrointestinal disturbances. In the dermatoses with gastrointestinal intoxication calomel and blue mass are among our most useful remedies. In recent years there has been a tendency to use small doses of mercury in the treatment of chronic inflammatory dermatoses for its direct effect upon the skin, in the same way that arsenic is used in the same sort of affections. In this way it is used in chronic thickened eczemas and other chronic inflammatory dermatoses; in lichen planus it is widely used.

The Iodides.—Potassium iodide, and to a much less extent sodium iodide and strontium iodide, perform an invaluable service in their specific effects upon the gummatous lesions of syphilis; and this is the chief use of the iodides in dermatology. They are also used for specific effect with great benefit in actinomycosis and blastomycosis. In addition to their specific uses, the iodides are widely used for their alterative effects. The salts of iodine stimulate tissue changes, and apparently it is this fact which renders them useful in a good many dermatoses. They are used with more or less positive benefit in scrofuloderma and other strumous affections, lupus erythematosus, gouty eczemas, and psoriasis. When administered for other than syphilis, actinomycosis, and blastomycosis, they are used ordinarily in small doses, one to five grains three times a day. Haslund has recommended the iodides in enormous doses for the treatment of psoriasis, but the digestive disturbances and the other distressing results of such doses render the treatment, even if efficacious, impracticable. In general it may be said that the usefulness of the iodides in syphilitic granulomata especially, and to a less degree in actinomycosis and blastomycosis, is of incalculable value, and that their use in other dermatoses is of relatively small importance. In view of the uncertainty of their results and on account of the disturbances of digestion and the other difficulties which arise from their administration, the iodides are little used except in the treatment of the first-mentioned group of diseases.

Arsenic.—Arsenic belongs to the small class of internal remedies which have a specific effect upon the nutrition of the skin. This effect is especially upon the cells of the epithelial layer, and consists, speaking broadly, of a stimulation of the nutrition of the cells. If the effect of arsenic is carried to an extreme degree there occurs, as shown by Ringer, Murrel, and Nunn many years ago in experiments on frogs, a degeneration of the cells of the prickle-cell layer so that the whole epidermis breaks down and readily peels off. When the action is not so acute but is long continued, as in arsenical poisoning, there occurs, not a complete degeneration of epithelium, but a stimulation of the nutrition of the cells, which is followed by an increased production of horny epidermis and of pigment. At times, also, there is observed in man, as a result of the ingestion of arsenic, hyperæmia of the skin with exfoliation of epidermal scales. In a general way this action may be compared to the effect which occurs in the epidermis as a result of active hyperæmia of the corium, whether this is produced by internal causes or by external irritants. And this comparison may serve to illustrate the commonest indication for its administration, viz., dermatoses in which we use external remedies for the purpose of stimulation. In other words, it is used in chronic inflammatory dermatoses in which we wish to cause solution and absorption of more or less well-organized inflammatory exudates. This action of arsenic upon the nutrition of the skin is also probably the reason for the benefit which has been found empirically to result from its use in certain dermatoses in which the lesions are due to disturbances of innervation.

On account of the first above-mentioned indications—its stimulating action we may call it—arsenic is of benefit in many chronic inflammatory dermatoses; in diseases in which, as result of the inflammatory process, there are thickened indurated lesions with more or less scaling. Such diseases are psoriasis, lichen planus, chronic thickened squamous eczemas —all diseases for which locally we use stimulating applications, like tar. Per contra, arsenic is not only not indicated, it is contraindicated, in acute hyperæmic diseases of the skin, especially in acute eczemas; or, to compare it again with external applications, in those conditions in which irritating local applications are contraindicated. According to the second of the above indications—its tonic effect upon the skin—arsenic is used with benefit in various dermatoses which are dependent upon disturbances of the nervous mechanism controlling the nutrition of the skin, like pemphigus, dermatitis herpetiformis, chronic urticaria, and disturbances of the sweat function. It is useful, also, perhaps for the same reason, in some acnes with sluggish skins and in acne produced by the bromides and iodides. It has been found empirically that arsenic is occasionally of use in retarding or destroying malignant growths. This action has been most definitely seen in sarcoma, but there is some ground for believing that it is of value occasionally in carcinoma as well, whether in the skin or more deeply situated.

In addition to its specific use in skin diseases, arsenic is given as a tonic in conditions where the general indications for such administration of it are found.

The most commonly used preparations of arsenic are arsenious acid, solution of potassium arsenite (Fowler's solution), and cacodylate of sodium. Other preparations, such as the bromide and iodide of arsenic, solution of sodium arsenate, arsenate of iron, are recommended, but offer no advantage over the commoner forms. There are some patients who have a marked idiosyncrasy to arsenic, and in all cases it is well to begin with small doses and gradually increase the dose, diminishing it immediately upon the appearance of puffiness about the eyelids, injection of the conjunctivæ, or gastrointestinal irritation. It should always be given after meals.

Phosphorus.—Phosphorus is a nerve stimulant, and is recommended where there are general indications for such an agent. In its action upon the skin it resembles arsenic, and is recommended in the dermatoses for which arsenic is used. It is a drug difficult of administration, and opinions of authorities as to its value in skin diseases are conflicting. It is usually administered in the form of zinc phosphide, dose $\frac{1}{10}$ to $\frac{1}{8}$ grain, phosphorated oil in capsules, dose the equivalent of $\frac{1}{100}$ to $\frac{1}{150}$ grain of phosphorus.

Salicylates.—Crocker recommends sodium salicylate and, preferably, salicin, to meet the same indications for which arsenic is used in chronic inflammatory dermatoses. He regards it as frequently more serviceable than arsenic, and recommends it in psoriasis, lichen planus, and other affections characterized by an inflammatory process of subacute intensity. He advises comparatively large doses—15 to 25 grains of salicin three times a day, after meals.

Quinine.—In dermatoses dependent upon nervous disturbances quinine in tonic doses is much used, and is generally regarded as serviceable. It is recommended in dermatitis herpetiformis and pemphigus, chronic urticaria and erythema multiforme, lichen planus, lupus erythematosus, and dermato-neuroses, like pruritus.

Antimony.—Malcolm Morris and Jonathan Hutchinson recommend the use of antimony in acute inflammatory disturbances of the skin, like acute eczemas. Its uses are thus exactly opposite to those of arsenic. It is given in small doses, from 3 to 10 drops of the wine of antimony three times a day, and is contraindicated by digestive disturbances. In acute hyperæmic dermatoses with itching, it has seemed to me to be of benefit at times.

Turpentine.—Crocker highly recommends turpentine for conditions similar to those for which wine of antimony is recommended; that is, acute hyperæmic disturbances, like acute eczemas and hyperæmic psoriasis. He thinks the method of administration is very important and recommends that it be administered as follows:

R	Oil of turpentine	♏x–xxx;
	Oil of lemon	♏ij:
	Mucilage of acacia, Water, } āā	℥ss.

This dose is to be taken three times a day, after meals, the last dos
to be later than 6 P.M., and during the treatment at least a qua
barley water is to be drunk during the twenty-four hours. These
cautions are in order to avoid irritation of the urinary organs fro
use of turpentine. An explanation of the usefulness of turpenti
such conditions as Crocker recommends it may, perhaps, be fou
its sedative effects on the cerebral and spinal centers.

Ichthyol.—Ichthyol was introduced by Unna and recommend
an agent for use especially against hyperæmia, both by local applic
and by internal administration. It and allied preparations, like thic
ichthalbin, are used to some extent in conditions in which there
indication for overcoming hyperæmia, as acne vulgaris, rosacea,
erythematosus. It is administered in doses of four to eight dr
capsules, three times a day after meals, and is, perhaps, at times u

Ergot.—Ergot, because of its action in causing contraction
muscular walls of the blood vessels, has been more or less used i
ditions characterized by hyperæmia or dilatation of the blood v
like acne, rosacea, erythema multiforme, and urticaria. Its va
problematical.

Pilocarpine.—Pilocarpine has a direct effect upon the skin.
action either on the cells of the sweat glands or on the nerve ter
in the cells it is a powerful stimulant of sweat secretion. Under it
tinuous use there may be also a stimulation of the growth of hai
doses just short of producing free diaphoresis, $\frac{1}{8}$ to $\frac{1}{12}$ grain of
carpine hydrochlorate, it is sometimes of great benefit in contr
itching in generalized acute eczemas, urticaria, pruritus, and
dermato-neuroses. It is a remedy that has to be used with circumsp

Atropine.—Atropine in its action upon the sweat glands
opposite of pilocarpine. In large doses it relaxes the terminal
vessels of the skin and produces an erythematous rash. It also de
the function of the sensory nerves and possesses slight anodyne
erties. It is sometimes used for excessive sweating and as a se
in urticaria and pruritus.

Thiosinamin.—Thiosinamin has the property of softening
tissue and is used hypodermically for this purpose. Its effect is u
temporary. Fibrolysin is a solution of thiosinamin and sodium sal
which is painless. It is furnished in tubes containing in sterile so
three grains of thiosinamin (the maximum daily dose).

Sulphur.—Sulphur is, in the popular mind, a sort of cut
sarsaparilla to which all sorts of unauthenticated virtues are att
It is a useful mild laxative and as such is valuable, especially fo
dren. It is excreted by the skin, and is a mild cutaneous stimula
as a rule little can be expected from its internal administration

eases of the skin. It is recommended in disorders of sebaceous and sweat secretions, in rosacea, acne, hyperidrosis, and sweat eruptions generally. In the form of fresh calcium sulphide it was highly recommended by Ringer for furunculosis, and it has been very widely used in suppurative processes in the skin. I believe it is useless in furunculosis.

Tar and Carbolic Acid.—Tar and carbolic acid have been recommended as alteratives in psoriasis, eczema, and itching dermatoses. It is very uncertain that they have any specific value.

Animal Extracts. Thyroid.—On account of the marvelous effect which the administration of thyroid glands has on the skin in myxœdema, it and its preparations, like iodothyrin, have been tried in many dermatoses. It is recommended as useful at times in lupus, scrofuloderma, icthyosis, psoriasis, chronic intractable eczemas, and scars. I have seen it markedly beneficial in a case of eczema in a myxœdematous patient. Ordinarily, however, the indications for its administration are not clear and its benefit is uncertain. Its administration has to be guarded carefully. Thyroid is most conveniently administered in the form of tablets of the desiccated gland, the dose of which to begin with should not be more than 5 grains daily, and it cannot be carried safely above 15 grains daily if the patient is not in bed. Symptoms of thyroidism must be watched for: flushing, giddiness, mental depression, palpitation, and rapid pulse.

Adrenalin.—Adrenalin has the property of causing contraction of the capillaries, and is both a local and general hæmostatic. It increases blood pressure, is a cardiac stimulant, and increases the urinary flow. It is comparatively harmless, and its use may be long continued without bad effects (MacGowan). As a local hæmostatic a 1 : 1,000 or a 1 : 5,000 solution may be applied on gauze; 15 to 30 c.c. of 1 : 1,000 solution on gauze may be safely packed in a wound. MacGowan[1] highly recommends adrenalin solution in angio-neurotic disturbances, like erythema multiforme, urticaria, and purpura. He gives it in doses of 10 gtt. of 1 : 1,000 solution every two hours by the mouth or hypodermically. It may thus be continued for forty-eight hours, and in smaller doses he frequently uses it for days or weeks. In a cachectic purpura in a 3½-pound baby he gave with excellent results ½ gt. of 1 : 1,000 solution every two hours for a week. The danger of producing arterial degeneration from the use of large doses or from the long-continued use of adrenalin should not be forgotten.

Ovarian extracts, like oöphorin, have been recommended in rosacea, and eczema occurring at the climacteric, and extract of the suprarenal glands for vitiligo.

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[1] *Jour. Cut. Dis.*, February, 1905.

Tuberculin.—Tuberculin may be used in dermatology as it is in other departments of medicine for diagnostic and therapeutic purposes. For therapeutic purposes it is used by hypodermic injections. It has had a wide trial in lupus and other forms of tuberculosis of the skin without satisfactory results, and it is now little used. For diagnosis in suspected cases of tuberculosis the hypodermic use of tuberculin gives information of definite value, but is not without danger. Because of this danger local tuberculin reactions are used for diagnostic purposes. There are four tests of this sort: The Von Pirquet, the Lignieres, the Moro, and the Calmette and Wolf-Eisner. The Von Pirquet is a vaccination test, using one drop of tuberculin and producing a scarification by twisting a sharp instrument, like a chisel. The Lignieres consists in rubbing in the tuberculin solution. The Moro test is an inunction of one to one and one half grains of an ointment of equal parts of old tuberculin and anhydrous lanolin. The Calmette is an ophthalmic reaction produced by placing in the eye one drop of a one per cent or one and one half per cent tuberculin solution. The Von Pírquet test has the greatest use, but because of its extreme sensitiveness and of the almost universal existence of small foci of tuberculosis in adults it is of little value except as a test of tuberculosis in children. In children under four years it is a reliable test. The Moro test is less sensitive than the Von Pirquet and is regarded as of greater use in adults for that reason. The Calmette test is not without danger to the eye and may produce permanent damage where an intense reaction occurs. All in all, where a tuberculin test is necessary in order to determine the character of a cutaneous lesion, a carefully carried out injection of tuberculin is the most reliable. The technique of these tests is given in various works on general medicine.

Opsonic Therapy.—Sir A. E. Wright has instituted a method of treatment of infections by the use of hypodermic injections of sterilized cultures of the bacteria. The principle underlying this method consists in raising, as a result of the stimulation produced by the vaccines, the amount or efficiency of the protective substances normally present in the blood serum to a point where destruction of the infecting organisms will result.

In infections nature's method of defense is by phagocytosis. The phagocytic cells, however, are unable to ingest the bacteria unless the latter have been influenced by certain substances in the serum, which Wright has named opsonins. Wright has shown that the amount of these opsonins in the serum of infected individuals is less than in the normal individual, and that by a course of injections of vaccine properly carried out, it can be raised above the normal. The ratio between the amount of opsonins in the serum of a given individual, and the amount present in the serum of a normal healthy individual, he has termed the opsonic index. This is determined as follows:

Since the opsonins are a property of the serum it is immaterial whether the leucocytes of the patient or of a normal person are used. In order to obtain leucocytes the blood of an individual is drawn into a 1 per cent solution of sodium citrate in 0.85 per cent salt solution, which prevents clotting. This is placed in the centrifuge, the supernatant fluid removed with a pipette, and the corpuscles, among which are the leucocytes, are washed free of serum. This mixture is thereby resolved into normal leucocytes (ignoring the erythrocytes) and normal serum. Serum from the patient is obtained in a similar manner; his leucocytes may be used instead of the normal leucocytes, if desired. Similar amounts of the two serums are then added to definite quantities of a staphylococcus emulsion which contains a suitable number of bacteria. To each of these tubes is then added identical quantities of washed leucocytes, the mixtures incubated for fifteen to thirty minutes, and at the end of this time the average number of cocci contained in the leucocytes of both tubes is determined after staining. The ratio between the two averages is the opsonic index.

In preparing the vaccine to be injected, bouillon cultures of the staphylococcus are made and incubated for three weeks at 37° C. This is then sterilized at 65° C., for from twenty minutes to an hour, and one half per cent carbolic acid or lysol added. Of this 1 c.c. is an average dose. The dosage is regulated more accurately, however, on the basis of the number of cocci injected rather than on the volume. The number of cocci in unit volume of culture is measured indirectly as follows: A mixture of equal quantities of the sterilized culture and of normal blood is stained on a slide and the ratio of cocci to blood cells determined. One may easily compute from this ratio the number of cocci in a cubic centimeter of the culture. The average emulsion contains from 2,500 to 5,000,000,000 of cocci to the cubic centimeter.

In the case of the tubercle bacillus, Wright uses Koch's tuberculin T. R., taking, however, the precaution of sterilizing it for an hour at 60° C. The blood of the patient is tested as to its opsonic powers, and the progress of the case is regularly controlled by subsequent similar determinations. The first effect of the administration of the vaccine is a temporary diminution in the antibacterial (phagocytic) power, which Wright calls the negative phase. This endures for a varying time, according to the individual and the amount of vaccine injected, and is followed by a stimulation of the formation of opsonins resulting in an increase of the antibacterial power, which as a rule rises for several days, decreasing again, however, to a point somewhat above the starting point. The injection of vaccine is then repeated.

Wright attributes his few failures in the use of this method to a neglect to determine the extent of this negative phase, the negative phases from the series of infections overlapping each other, and thus causing a decrease in the opsonic index. He speaks of this as a cumulative phase. By proper dosage and proper spacing of the injections the negative phase may be eliminated largely, and a cumulative positive phase established. The latter is represented by a high opsonic index. Wright and others have reported favorable results from the use of this method in various pus infections of the skin, including furunculosis, sycosis, and acne, and in lupus vulgaris. Larger experience is showing the method to have only a limited field of usefulness.

The list of agents enumerated above is fairly comprehensive of the remedies which are used internally for their direct effect upon the skin. A consideration of them is not likely to impress one with the great importance of specific internal medication in the treatment of skin diseases. It rather brings us back to the proposition that the successful treatment of diseases of the skin, in so far as it consists in the use of internal remedies, must depend upon rational, as distinguished from specific, therapeutics.

EXTERNAL TREATMENT

Varieties of External Applications

Upon the basis of their therapeutic effects, most of the local applications used in dermatology can be divided into the following groups: (1) detergents, (2) soothing and protective applications, (3) antipruritic applications, (4) anæsthetics, (5) stimulating applications, (6) caustics, (7) keratolytic agents, (8) keratoplastic agents. A brief consideration of these groups is useful for the purpose of comprehensive orientation, and especially for the purpose of indicating the principles of action upon which the use of local applications depends.

Detergents.—Detergents are used to remove the grease and scales of the skin, pus, crusts, and extraneous matter. Water, alone or with soap and various other substances, is used in the form of baths, wet dressings, and lotions. Bathing or washing is usually irritating to acutely inflamed surfaces; such surfaces may be cleansed at times by the application of wet dressings, which are less irritating because of their cooling effect and the protection from the external air that they afford. When it is undesirable to use water, surfaces can be cleansed with oils and thin neutral fats.

Soothing and Protective Applications.—The general purpose of soothing applications is to relieve irritation. This may be done in various ways: by protective applications; by applications which from their evaporation cool the surface; by dilute astringents which tend to overcome hyperæmia; and by agents which have a positive sedative effect upon the nerve endings. The first three forms of these are curative; they relieve symptoms by influencing the pathological process. The fourth form—agents which have a sedative effect—may not be curative, may in fact be harmful, only relieving symptoms by dulling sensation. Soothing applications are indicated in all sorts of conditions of irritability of the skin, but especially in acute inflammatory diseases. Simple protective applications, while they have only the negative property of preventing irritation, are often all that is necessary in order to allow a sensitive surface to become quiet. In furnishing protection to an acutely inflamed cutaneous surface we have to bear in mind protection not only against dirt and traumatism, but more especially against the irritation which comes from contact with external air. Protection is furnished by the use of inert powders, by the application of ointments and similar preparations, by wet dressings, and, less commonly, by other forms of covering. Agents which reduce irritation by a cooling effect owe this property generally to the presence of water, which evaporates during their application. The cooling agents used in acute inflammatory conditions all depend largely upon the evaporation of water. Volatile substances like alcohol, which are irritating to the broken skin, may be used, usually in dilute solutions, upon unbroken surfaces, and owing to their quicker evaporation cool more rapidly than water. To cause contraction of the injected capillaries in acute inflammatory conditions, and in that way to counteract the irritation of an inflammatory process, very dilute astringents are used, such as subacetate of lead, acetate of aluminum, ichthyol. For direct sedative action upon the nerve terminals in the skin such agents are used as opium and carbolic acid in very dilute forms.

Of course we often combine all of these indications in one application, or indeed one agent may meet all of the indications. Thus, for example, a mixture may be made as follows:

Tr. of opium, Sol. of subacetate of lead,	āā	℥ss.;
Zinc oxide		℥ij;
Water	ad	O.i.

In such a mixture the opium and the lead salt are astringent and sedative; in the evaporation of the water there is produced cooling of the surface which is soothing and also tends to constringe the blood vessels; the inert zinc oxide powder which is deposited upon the surface is slightly astringent, but chiefly furnishes mechanical protection and at the same time increases evaporation from the surface.

Antipruritic Applications.—The soothing applications are antipruritic, because itching is the chief manifestation of irritation the skin. In addition, however, to the soothing applications there other agents which are used distinctly for their antipruritic acti These antipruritics may act either by producing a direct sedative eff upon the sensory mechanism of the skin, or by producing as a sub tute another sensation in the skin. An example of the first group carbolic acid, which is distinctly anæsthetic to the skin. Of the age which relieve itching by substituting another sensation for itchi menthol, which acts by producing a sensation of cold, is an exam Nature furnishes in scratching an instinctive method of relieving it ing by substituting another sensation. Unfortunately its effect is t porary, and the traumatism which it produces is the cause of m damage in itching dermatoses.

Anæsthetics.—Agents for producing anæsthesia in the skin are used frequently, because the indications for producing complete an thesia in the skin are fortunately not of frequent occurrence, and w they do occur cannot readily be met. For partial anæsthesia carb acid in various strengths is used; for more complete anæsthesia, coca eucaine, and orthoform. For evanescent complete anæsthesia rapi evaporating liquids, which can be sprayed upon the surface like liq ethyl chloride, are used. These by their rapid evaporation freeze surface and render it momentarily anæsthetic.

Stimulating Applications.—These are used chiefly to produc greater or less inflammatory reaction in order to cause the absorpt of pathological products in the skin. Their chief indication is for removal of the partially organized exudates which result from persist inflammatory processes of low intensity. Stimulants are indicated in sluggish wounds to render the reparative process more active. stimulating applications used in dermatology are nearly all dilute s tions of strong irritants or caustics. Among those in common use tar, sulphur, mercury and its salts, especially the bichloride, green s silver nitrate, carbolic acid, iodine and its compounds like iodofo balsam of Peru, and chrysarobin. Most of these irritants largely dil are antipruritics.

Caustics.—Caustics range in intensity from irritants which stroy the epidermal cells with which they come in contact and ca their exfoliation, to those which penetrate deeply, destroying all sues. Among those of weaker intensity in common use are salic acid, bichloride of mercury, tincture of iodine, resorcin, must capsicum, cantharides, and nitrate of silver. To produce superfi caustic action in the corium when its tissues are exposed, nit of silver and carbolic acid are the agents of commonest use. T are never to be relied upon, however, when deep destructive effect is necessary, for the coagulation of albumen which they produce prevents their deep penetration. Stronger caustics are glacial acetic acid, trichloracetic acid, chromic acid, and sodium ethylate (which must be fresh). Strong caustics which will destroy the epidermis and penetrate deeply are the common mineral acids, of which nitric acid is the most frequently used, acid nitrate of mercury, arsenic, zinc chloride, caustic potash. These latter are only used when it is intended to produce immediate destruction of circumscribed masses of tissue, like warts, moles, and small malignant growths. In the attempt to cause the immediate destruction of lesions like warts, which are covered with horny epidermis, one is apt to be surprised by the resistance which the horn cells offer against destruction. So great is this that where it is desired to cause destruction of tissue by any but the most powerful caustics it is advisable first to dissolve off the horny epidermis by moderately strong solutions of caustic potash or zinc chloride. Such caustics as nitric acid, zinc chloride, and potassium hydrate destroy very rapidly any tissue with which they come in contact, and their action, therefore, has to be guarded. Of these caustic potash is the most destructive and penetrates most deeply.

Keratolytic Agents.—Keratolytic agents are those which cause solution of the horny epidermis; their action is, of course, chemical. Salicylic acid is the best illustration of these, and is the one which is ordinarily used when one wishes to remove masses of horny epidermis. Soaps containing free alkali are also used. Many substances, as, for example, boric acid, are keratolytic to a slight degree. The alkaline caustics are all markedly keratolytic, and for the rapid solution of horny epidermis their dilute forms are used, as, for example, liquor potassæ. For more extensive applications, especially when it is desired to remove large accumulations of horny scales, use is ordinarily made of green soap, which owes its keratolytic action to the free potassium hydrate contained in it.

Keratoplastic Agents.—Keratoplastic agents are agents which tend to hasten the conversion of soft epidermal cells into horn cells. The method of their action is not entirely settled, and probably varies with different agents. Unna has attempted to explain the therapeutic properties of a large number of agents which in their weaker action are keratoplastic by classifying them as reducing agents in the chemical sense, that is, agents which abstract oxygen from other substances. According to his theory, such agents have a superficial and a deep effect; in their weaker action they promote cornification by abstracting oxygen from the epithelial cells. In their stronger action they abstract oxygen not only from the epithelial cells, but also from the endothelial cells of the superficial blood vessels as well. As a result the endothelial cells shrink, the vessels are diminished in caliber, and the blood supply is diminished. They are accordingly antiphlogistic. Further, they are antiparasitic against organisms in the skin because they deprive them of their supply of oxygen. Among weak reducing agents he includes ichthyol, thiol, sulphur, and the quicksilver salts; among the stronger, tar, aristol, resorcin, pyrogallic acid, and chrysarobin. Unna's theory of reducing agents has had wide attention and it is an ingenious application of a chemical principle, but that the property of his reducing agents of abstracting oxygen from the tissues is the explanation of their therapeutic properties is not clear. That the abstraction of oxygen from pathological cells causes their more rapid cornification may be true, and may well be the explanation of the action of certain keratoplastic agents, but that the diminution in the oxygen supply of the endothelium of the capillaries is the explanation of the shrinking of the blood vessels is open to weighty objections. The same is true to a less extent of his explanation of the parasitic action of his reducing agents. Most of his reducing agents are irritants—in fact in concentrated forms they are keratolytic agents and are only keratoplastic when in dilutions—and Leredde suggests that the explanation of their action rests in the defensive reaction of the tissues which they produce.

It may well be that cornification is hastened by agents which abstract oxygen from the epithelial cells, but that action is probably only one of several that promote cornification. It is not unlikely that the abstraction of liquid from a raw epithelial surface will hasten cornification of the cells, and from what we know of the effect of pressure and friction in favoring the production of horn cells, it is probable that substances that exert slight pressure upon the soft epithelial cells will have the same effect. Thus the keratoplastic action that is noted from the application of various powders upon raw epithelial surfaces may be due partly to their drying action and to the mechanical pressure exerted by the thin layer in which they cake. Further, all remedies which are classed as astringents, whatever their methods of action, are probably more or less keratoplastic.

We do not frequently have to avail ourselves of keratoplastic agents as such; the cells undergo normal cornification with sufficient rapidity if the underlying pathological conditions are removed. The keratoplastic remedies ordinarily used are those enumerated above as the reducing agents of Unna.

Antiseptics.—In dermatology, as in general surgery, asepsis is better than antisepsis, and where noninfected lesions of the skin occur one always aims to avoid infection if possible. But pus-forming organisms are so ubiquitous that only the greatest care in protecting open lesions from contamination will prevent infection unless antiseptic applications are used. Indeed infection with pus organisms is the rule in lesions of the skin in which the horny layer is lost, unless the lesions have competent care from their appearance. The vigor with which antiseptic applications have to be applied varies considerably under different circumstances. In acute infected dermatitis the use of strong antiseptics is impracticable and fortunately they are not required, for the applications of the blandest wet dressings, such as boric-acid solution or normal salt solution, by providing thorough drainage, are sufficient to clean up the surfaces. In the treatment of infections in extensive open surfaces of the skin, wet dressings are the most satisfactory because they provide for drainage. In treating small suppurative surfaces or in protecting against infection of larger open surfaces, ointments containing antiseptics are often satisfactory, but are open to the objection that they do not allow drainage. In infective processes deep in the skin beneath an unbroken epidermis, the external application of antiseptics is useless, for the unbroken horny layer presents an effectual barrier to their penetration. Antiseptics in the form of powders are also used on suppurating surfaces and especially on ulcers, but if the secretion is abundant they are apt to be unsatisfactory because they cake and form crusts under which the secretion accumulates. The antiseptics used in dermatology are those commonly used in general surgery.

Parasiticides.—There is frequent demand for the use of parasiticides against the animal and vegetable parasites which attack the skin. Those in commonest use are sulphur and its compounds, especially sodium hyposulphite and potassium sulphide, the mercurial salts, styrax, Peruvian balsam, naphthol, and chrysarobin.

Local Remedies

The local remedies which are used in diseases of the skin are very many. It is not practicable to consider all of them, nor is it desirable, for the properties on account of which they are used in dermatology are those which are described in general works on materia medica and therapeutics. A few of them, however, are entitled to consideration here, either because of the frequency of their use in dermatology or because of their typical character.

Boric Acid.—Boric acid is a weak disinfectant, and antiseptic and keratoplastic agent. These properties, however, are not sufficiently active to render it a valuable therapeutic agent, were it not also the fact that it is soothing in its effect. It is a common ingredient of mildly antiseptic soothing preparations, whether in the form of powders, lotions, or ointments. The pure, impalpable powder is not irritating, and can be dusted upon inflamed surfaces. It is soluble in twenty-five parts of water, and its saturated solution is an exceedingly useful mixture for cleansing and for application in the form of wet dressings. Boric acid, in the proportion of ten to twelve per cent, is an excellent addition to soothing ointments.

Salicylic Acid.—Salicylic acid is a disinfectant and antiseptic. When greatly diluted—one to two per cent—in ointments and powders and other forms, it is astringent and soothing and keratoplastic. In stronger proportions it is the keratolytic agent par excellence; it slowly but perfectly dissolves epithelium, loosening the epidermis from the corium with the production of very little inflammation. It is for this reason the chief ingredient of applications which are used for the removal of callosities and other horny hypertrophies. Salicylic acid is one of the very commonly used drugs in dermatology. In the proportions of one or two per cent it may be added with effect to many applications for acutely inflamed surfaces. In slightly stronger proportions, up to five per cent, it is used as an antiseptic, antiparasitic, and keratolytic agent in dermatoses which are not acutely inflamed. For the removal of horny masses it is used much stronger, from ten to twenty per cent. In such cases it is best combined with a vehicle like collodion, which limits its action sharply to the particular point.

Resorcin.—Resorcin in its therapeutic properties is very similar to salicylic acid. It is antiparasitic and antiseptic; in weak proportions —one to three per cent—it is astringent and keratoplastic and nonirritating; in strong proportions—ten to twenty per cent—it is keratolytic. It is a frequent addition to external applications in the proportion of one to three per cent for its antiseptic properties. In the proportion of ten per cent it is used on account of its keratolytic properties when it is desired to cause exfoliation of the epidermis. It has largely been used through Unna's recommendation as an antiseptic against the organism of Unna's seborrhœic eczema.

Sulphur.—Sulphur is a typical representative of Unna's reducing agents. It is antiseptic and parasitic; in dilute strength it is keratoplastic and astringent; in stronger proportions it is irritating and causes exfoliation of the epidermis. Sulphur is itself inert, but on the surface of the skin some of it is converted into hydrogen sulphide and upon raw surfaces there are formed from it sulphurous and sulphuric acid, and its therapeutic properties are in large part or wholly due to these changes. In the strength of two to four per cent it can be used upon inflamed surfaces without producing irritation. For its stimulating and parasitic effect it is used much stronger, ten to fifteen per cent, but such strengths are apt to produce dermatitis. Sulphur is a peculiarly useful antiseptic and parasiticide in skin diseases, and is one of the very frequently used agents. Sulphur is official in the form of sublimed sulphur, precipitated sulphur, and washed sulphur. Precipitated sulphur, because of its greater purity, is the preferable form for local applications.

One of the greatest disadvantages in the use of sulphur is that it is insoluble. To overcome this objection a colloidal sulphur has been introduced which is soluble in water. For most purposes it may be used in strengths of five to ten per cent in water or ointments. The indications for its use are the same as those for sulphur.

Ichthyol.—Ichthyol has the therapeutic properties of sulphur. It is a strong reducing agent; it is antiseptic and antiparasitic; in dilute strength it is keratoplastic and astringent; pure it is slightly irritating. It was introduced by Unna, and has been especially recommended for its property of causing, both by its local application and by its internal use, diminution in the caliber of dilated capillaries. It has accordingly been largely used both in inflammatory conditions for its astringent effect, and in other pathological processes with dilated blood vessels. It is a valuable astringent much used in acute inflammatory processes, because it is not irritating except when in strong proportions. Its dirty appearance and bad odor are objections to it.

Mercury and its Salts.—Metallic mercury in the form of mercurial ointment is commonly used by inunction for its specific effect in syphilis. Mercurial ointment is also a commonly used but dirty and objectionable parasiticide. Aside from their specific use in syphilis the salts of mercury are among our most valuable local therapeutic agents in dermatology, because of their antiseptic, antiparasitic, and stimulating properties. In dilute proportions they are slightly stimulating to the skin, and, like other stimulants, where their action is very weak they are soothing and antipruritic. In strong proportions they excite dermatitis and cause exfoliation of the epidermis. The salts of mercury most commonly used in dermatology are the bichloride, ammoniated mercury, and calomel. The bichloride is too irritating for use in irritable raw dermatoses, but in very dilute solutions, 1:10,000 to 1:5,000, it is, in the form of wet dressings, one of our most useful antiseptic applications in suppurating wounds of the skin. The bichloride in strong solutions, 1:1,000 to 1:200, is used in certain conditions for the purpose of producing exfoliation of the epidermis. Ammoniated mercury, two to ten per cent, in the form of ointment is much used as an antiseptic and local stimulant. Ammoniated mercury, three to five per cent, in ointment is an efficient and nonirritating application for small areas of suppurating dermatitis. In stronger proportions, eight to twelve per cent, it is a useful stimulating ointment for exciting a healthy inflammatory reaction, as in psoriasis. The properties of calomel are similar to those of ammoniated mercury, and it is used for the same purposes. Mercury and mercurial salts when applied in the form of ointments are readily absorbed by the skin, especially if they are rubbed in, and care therefore must be taken to avoid their too extensive application.

Iodine and the Iodine Preparations.—Iodine is a penetrating antiseptic and parasiticide, and is a violent irritant to the skin. In the form of the tincture it is much used when it is desired to produce an acute inflammatory reaction in the skin and to cause the deep destruction of bacteria. Its effect upon bacteria below the epidermis is probably produced through the inflammatory reaction which it excites rather than by any direct action upon the bacteria. Numerous synthetic compounds which depend upon iodine for their antiseptic properties are in use in dermatology, like iodoform, aristol, europhen, iodol. In its peculiar way iodoform is the most valuable of these. After iodoform, and without the disadvantage of a penetrating disagreeable odor, aristol is perhaps the most useful. It is nonirritating, and is a satisfactory antiseptic and keratoplastic agent.

Carbolic Acid.—Carbolic acid is antiseptic, antipruritic, and anæsthetic. It is used as an antiseptic in dermatology, as in general surgery, but its chief use in dermatology is on account of its benumbing effect upon the sensory nerves of the skin. It is the cutaneous anæsthetic, and is the most frequently used antipruritic remedy. It is effective against itching, not only in inflammatory dermatoses, but when the skin is apparently normal. For its antipruritic effect carbolic acid is used in the strength of one to five per cent. Carbolic-acid solutions when applied continuously in wet dressing may produce gangrene of the skin. This may occur even from wet dressings of one per cent solution. Because of this danger, wet dressings of carbolic-acid solution are not safe and should be avoided. Pure carbolic acid is destructive of living tissues, but it is not a very penetrating caustic. A single application of the pure acid or of a very strong solution may be made to small areas of the skin for anæsthetic effect or for destruction of organisms, but, on account of the possibility of necrosis, such an application should not be repeated until the surface has entirely recovered from the previous application.

Tar.—In dilute strength tar is antipruritic and astringent. In stronger proportions it is a stimulant or irritant. Tar is the type of stimulating applications which are used to produce a healthy reaction in subacute inflammatory dermatoses. It is a particularly useful stimulant in such conditions, and this, in connection with its antipruritic action, brings it into constant requisition. It is one of the most useful of the dermatologist's remedies, but its proper use requires more than ordinary care. Tar is the remedy with which the dermatologist usually passes from soothing to stimulating applications in the stage of subsidence of inflammatory dermatoses. In taking this step it is wise, in order to avoid harm, to proceed very cautiously, and at first to use very dilute preparations (two to four per cent), and to increase the strength tentatively. As an antipruritic in acute inflammatory conditions a strength of two to four per cent may be all that can well be used. As a stimulant in chronic inflammatory dermatoses it is used in strengths of from six to twenty per cent, and occasionally where it is desired to produce a strong reaction a vigorous application of the pure tar may be used. Tar is a very complex substance, containing oil of turpentine, creosote, resins, and various other ingredients, and according to its origin, varies somewhat in its composition. In dermatology we use pix liquida (wood tar), oleum cadini (oil of juniper), and oleum rusci (oil of birch). These do not differ materially in properties, but pix liquida and oleum rusci are apt to be the most reliable preparations in America.

Tar is ordinarily used with an ointment or paste as the vehicle. It is very sparingly soluble in water, and therefore cannot be readily used in lotions. To provide for its use in aqueous solutions several liquid preparations of tar have been suggested; the best of these are Bulkley's liquor picis alkalinus and Duhring's compound tincture of coal tar. Bulkley's formula is as follows:

Pix liquida	ʒij;
Potassa caustica	ʒi;
Aqua	f℥i.

To make Duhring's compound tincture of coal tar, one part of coal tar should be digested in six parts of tincture of quillaja with frequent agitations for at least eight days. The product is a brownish-black tincture which in water forms a yellowish emulsion. Both of these are used largely diluted—in strengths of from one to ten per cent.

Chrysarobin.—Chrysarobin is antiseptic and antiparasitic and a powerful irritant to the skin. After tar it is, perhaps, the most typical of the stimulating applications which are used in dermatology. It is a more powerful stimulant than tar and must be used with correspondingly greater caution, except in dermatoses where the production of an acute inflammatory reaction is not especially to be dreaded. Chrysarobin is a valuable agent in the treatment of fungus diseases of the skin, but its especial use is in the treatment of chronic inflammatory dermatoses to produce a healthy reaction. In certain dermatoses like psoriasis, the reaction excited in the lesions is apparently much less than that in the surrounding skin. Chrysarobin is used in the strength of from two to ten or even twenty per cent; in weaker proportions it is often quite as effective as when used stronger, and it does not then excite the acute dermatitis which accompanies its use in greater strengths. In addition to the irritation which it produces, chrysarobin has the great objection of staining everything with which it comes in contact. It stains the skin a brownish color, the hair a greenish yellow, and the nails a reddish brown, and these stains disappear rather slowly. On account of the staining of the hair and of the conjunctivitis which it is liable to cause when used upon the face, it cannot well be used about the head. It also stains clothing a walnut brown, but this can be removed by dilute solution of caustic soda or solution of chlorinated soda. In spite of the drawbacks to its use, chrysarobin is so valuable a remedy that it cannot be dispensed with. It is ordinarily used with ointments. In order to limit the extent of its application and to avoid staining the clothing, it is frequently employed in the form of a varnish, like the following:

Chrysarobin	1 to 10 parts.
Solution of gutta percha (traumaticin)..ad	100 "

This is applied in the same way as collodion and dries upon the surface, leaving a protective covering.

Pharmacology

The principal forms in which external remedies are applied in dermatology consist of powders, lotions and other liquid applications, ointments, and pastes. Certain other forms are less commonly used, as soaps, plasters, gelatine and similar dressings. All of these different forms have certain advantages under certain circumstances, and are worthy of consideration.

Powders.—Powders, when spread upon the surface of the skin, have certain properties which are dependent upon their physical form. In the first place they are convenient to use, and when they adequately meet the therapeutic demands they furnish an agreeable form of application. They afford a certain amount of protection to the surface. They are absorbent and at the same time promote evaporation, so that if secretion is scant they provide sufficiently for its dissipation. They are thus soothing and cooling to the surface, and this and the mechanical protection which they furnish are their most important functions. The cooling property of inert powders depends upon the fact that they promote evaporation from the surface, the explanation being as follows: The evaporation surface of the skin is practically a plane surface. When a layer of powder is applied to the skin—we will imagine for clearness that this is a single layer of particles—the surface is covered with a layer of approximately spherical bodies, over the surface of each one of which the secretion of the skin spreads. The evaporating surface is then represented, not by the plane area of the skin, but by the total area, or, admitting that evaporation only occurs from the upper hemisphere of each spherule, by one half the total area of all the spherules which cover the surface. In other words, the evaporating area is increased from two to four times. (Total area of surface covered is πR^2. Total area of spherules is $4\pi R^2$.) It is evident that if we want to provide for the evaporation of only a very slight amount of secretion, as, for example, insensible perspiration, a very thin layer of powder is sufficient. If the secretion, however, is rather abundant, a thicker layer of powder should be used in order to provide for its more rapid absorption. The disadvantage which renders powders a not very effective means of external application is that the protection which they furnish is only slight, and they are easily rubbed off, and if a secretion is abundant they cake upon the surface and form masses which are mechanically irritating. To avoid irritation it is important that powders should be impalpable.

The ordinary method of applying powders is to dust them over the surface. In order to get a continuous effect from them, Unna has suggested their application in bags. The bags are made of a loose-meshed gauze and quilted in opposite directions, so that the powder cannot shift about. These bags are bound upon the surface. Occasionally this method of applying powder is useful.

The almost inert powders which are used upon the skin are numerous. Among the most common are zinc oxide, zinc carbonate, and zinc stearate, calamine, magnesium carbonate, bismuth subnitrate, talcum, kaolin, prepared chalk, starch, orris root, lycopodium. Of these the oxide of zinc is the most commonly used. Stearate of zinc is a very light powder of greasy feel which adheres readily to the skin, forming a layer which does not prevent the exit of liquids, but protects against their absorption from without. Calamine is an impure carbonate of zinc of a salmon-pink color, and is useful in mixtures with white powders to make a flesh color. With equal quantity by weight of zinc oxide it makes a pinkish powder which is hardly noticeable on the skin. Magnesium carbonate is very light and is the most absorbent of these powders. The vegetable powders, especially starch, have the objection of caking readily when moist, and of decomposing. Lycopodium, which is a pollen, is not so objectionable in this particular. According to Gruendler[1] the relative absorbent power of powders is as follows: Magnesium carbonate will take up 5½ times its weight of water; infusorial earths and siliceous substances 3½ times their weight of water; kaolin, 1½; zinc oxide, 1⅕; starch, 1; talcum, ⅗; lycopodium, ½; prepared chalk, ⅖.

In addition to the inert powders which are applied for their physical effects, certain powders, usually largely diluted with inert powders, are applied on account of their medicinal action. Such are antiseptics, like boric acid, salicylic acid, iodoform, aristol, camphor, calomel, carbolic acid, orthoform. In using powders merely for their physical effect, it is often desirable to mix them; for example, we get a powder of good weight by mixing zinc oxide, which is heavy, with starch, which is light, or by mixing zinc oxide with talcum or with lycopodium and starch. If a powder is to be used upon the face it is very desirable to have it of a flesh color, like the mixture of zinc oxide and calamine referred to above. Another flesh-colored powder suggested by Unna has the following formula:

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[1] *Monatshefte*, No. 20, 1888.

℞ Zinci oxidi	2 parts;
Magnesii carbonatis	3 “
Boli albæ	3 “
Boli rubra	2 “
Amyli oryzæ	10 “

To all of these inert powders it is a good plan to add a nonirritating quantity of some antiseptic; boric acid, in the strength of about ten to twenty per cent is the most frequently eligible addition. Salicylic acid (one to two per cent), acetanilid (five to ten per cent), are used in the same way. Carbolic acid (one to two per cent), camphor (five to ten per cent), are used as antipruritic powders.

We have in orthoform an anæsthetic powder which is nonirritating upon application, and is said to be nonpoisonous. In the proportion of ten per cent mixed with other powders, as boric acid, it is anæsthetic, but it has the great objection that it frequently causes a violent dermatitis, and sloughing of the skin may occur from its use. It is, of course, not absorbed by the unbroken epidermis, and is only anæsthetic upon denuded surfaces.

An excellent way of getting the effect of powders is by suspending insoluble powders, like oxide of zinc, in lotions. When these are applied to the surface and evaporation takes place the powder is left in a thin, smooth layer. Such mixtures will be considered under lotions.

Glycerine.—Pure glycerine is exceedingly hygroscopic, and when rubbed upon the skin it abstracts water rapidly and is irritating. When largely diluted with water, however, it acts like the fats, and in the proportion of two to ten per cent in water is soothing. It is a common addition to lotions to give them body and to prevent their too rapid drying. In order to still further increase these properties one may add a few grains of tragacanth with glycerine, 1 to five grains to the ounce.

Baths.—The application of water to the skin produces both physical and chemical effects. The mere contact of water with the skin, especially if accompanied by motion, produces a certain amount of stimulation. But the chief physical action of water upon the skin is thermal. Either cold or hot water applied to the skin causes temporary contraction of the capillaries, which is subsequently followed by their dilatation. The application of tepid water causes an immediate dilatation of the cutaneous capillaries. The action, therefore, of water of any temperature which appreciably varies from the temperature of the skin produces either primarily or secondarily a transitory hyperæmia and is stimulating. The chemical effect of water upon the skin is that of a detergent. It dissolves dirt and excretions upon the skin, and causes a certain amount of maceration of the horny epidermis. Where the horny layer is unbroken this effect upon the skin is minimal, but there is even then some absorption of water by the horny cells and a certain amount of maceration. That this is not greater is due to the fats which infiltrate the horny layer, and when solvents of the fats, like soaps and the alkalies, are dissolved in the water this effect is greatly increased. Where the horny layer is absent water is taken up freely by the deeper cells of the epidermis, causing them to swell, some of them to the point of rupture, and upon such surfaces water is an irritant. It may be readily seen from the foregoing that the application of water to the skin in dermatoses is not a matter of indifference. Too frequent bathing, especially with soap, by depriving the skin of its fatty protection increases its vulnerability, while the hyperæmia which follows bathing tends to exaggerate any inflammatory process which may exist.

Baths, according to their temperature, are classified as follows:

Cold baths	from 40° to 65° F.
Cool baths	from 65° to 80° F.
Tepid baths	from 80° to 95° F.
Warm bath	from 95° to 100° F.
Hot baths	from 100° to 110° F.

Cold and cool baths are stimulating to the nervous system, and may be used for their general physical effects, and in certain dermatoses they may be valuable in stimulating a sluggish skin. They tend to make the skin firmer and to improve its texture. Both these and other baths are contraindicated where cutaneous irritants are contraindicated. To get the tonic effect of cold baths they should not be prolonged beyond a few minutes; they are distinctly depressant when overdone. Baths for the purpose of increasing elimination have to be somewhat prolonged, from five to twenty minutes. When extended to the duration of fifteen to twenty minutes they are depressant. Turkish and other forms of vapor baths are used especially for increasing elimination, and they may be used to advantage in certain dermatoses. The action of these baths is purely thermal, furnishing a convenient method for the application of heat to stimulate the circulation of the skin and the activity of its glan Such baths are also directly stimulating to the skin itself. This stin lating effect is increased by following the hot bath with a cold bath cold sponge bath.

The chief use of warm and hot baths in dermatology is for th stimulating and detergent effects, and for this purpose they are usu combined with alkaline soaps or other alkalies. They are not only u for cleansing purposes, but much use is made of them in chronic sc dermatoses or where there is extensive formation of horny epidermis the purpose of getting rid of the excess of horny scales. Such ba should be prolonged for ten to twenty minutes in order to produce mac tion of the epidermis.

The continuous bath was introduced by Hebra in the treatment certain dermatoses in which there is loss of extensive areas of epider such as pemphigus and burns. The continuous bath consists in mersing the patient's entire body except the head in a bath of body t perature, which may be continued uninterruptedly for weeks without apparent ill effects upon the health. The use of such baths is still ommended. The continuous bath, however, is difficult to carry out, in my opinion it is to be condemned except, perhaps, in extensive ca of certain types of pemphigus.

Medicated baths have a small field of usefulness in dermatol Aside from the effect of the water they furnish a convenient method the application of a few drugs in certain dermatoses. Medicated ba should be tepid or warm and of five to ten minutes' duration. They usually given as full-length baths, which are conventionally estima at thirty gallons of water. The most frequently used medicated ba with the preparations for thirty gallons of water are the following:

Emollient Bath.

Bran	2 to 5 pounds;
Starch	1 to 3 "
Gelatine	1 to 3 "
Linseed	1 to 3 "
Or size	1 to 3 "

Any of these materials should be allowed to steep for five to ten min before the bath is used. These baths are slightly detergent and are irritating. They are chiefly used as a substitute for plain baths when latter are irritating to the skin.

Alkaline Bath.

Sodium bicarbonate	℥ij to ℥x;
Or borax	℥ij to ℥x;
Or potassium carbonate	℥ij to ℥vi.

The alkaline bath is nonirritating, and may be used as a substitute for the plain bath when bathing is necessary in acute inflammatory dermatoses. It is also antipruritic, and is valuable for this effect in such conditions as urticaria and prurigo. It is used also for its detergent effect, but where a detergent effect is desired a more effective bath is made by one half to one pound of green soap.

Salt Bath.—Four to ten per cent sodium chloride. Stimulating.

Sulphur Bath.—This is most conveniently prepared with potassium sulphide, 2 to 4 ounces. Sulphur baths may also be made by using one ounce of Vleminckx's solution, the formula for which is as follows:

℞ Lime	℥ss;
Sublimed sulphur	℥i;
Distilled water	℥x.

Boil down to 6 ounces and filter.

Startin's sulphur bath is as follows:

℞ Precipitated sulphur	℥ij;
Sodium hyposulphite	℥i;
Dilute sulphuric acid	℥ss;
Water ad	O.i.

Mix and add to bath.

The sulphur bath is useful in scabies and in certain inflammatory dermatoses.

Acid Bath.

℞ Nitric or hydrochloric acid	℥i.

Mercurial Bath.

℞ Corrosive sublimate, Sodium chloride or hydrochloric acid, āā	ʒi to ʒiij.

As an antiseptic bath and for use in syphilodermata.

Neither the sulphur, acid, nor mercurial bath should be given in metal tubs.

Iodine Bath.

℞ Iodine	ʒss;
Potassium iodide	℥ss;
Glycerine	℥iij.

or

℞ Iodine	ʒss;
Liquor potassæ	℥i to ℥ij.

Bromine Bath.

℞ Bromine	gtt. xx;
Potassium bromide	℥ij.

Potassium Permanganate Bath.

Potassium permanganate	ʒiss to ʒij.

Antiseptic and sedative.

Menthol Bath.

℞ Five-per-cent solution menthol in alcohol	℥ss to ℥i to the bath.

All of these baths can be used as local baths as well as full baths.

Tar Bath.—This is given by anointing the surfaces with tar and then placing the patient in the bath for from one half to one hour. He is then washed off with soap and sponged off with cool water, and either powdered with an inert powder or anointed with fat or oil. It is only indicated when a violent stimulating effect is needed. Instead of using tar, the following mixture may be substituted:

℞ Oleum rusci, Oleum fagi,	āā	50
Spiritus vini rectificatus, Oleum olivarum,	āā	25

The natural thermal and mineral baths have the same indications as artificial baths of the same characters. They are usually sulphur baths or simply hot baths. Some of them give a temporary velvety feel to the skin, doubtless due to the presence of aluminum silicate or other silicates which they contain. There is no reason to believe that they possess any unique properties.

Wet Pack.—The stimulating effect of the cold bath may be gotten by wrapping the patient in a sheet wrung out of cool or cold water. Vigorous rubbing is given while the pack is continued, and after a few minutes the body is dried with vigorous rubbing. The warm wet pack may be used as a substitute for the warm bath for a detergent effect. The patient is first wrapped in a sheet wrung out of warm water, and then in a dry blanket, and left from twenty minutes to an hour. After this the skin is rubbed down to remove scales and crusts and exfoliating epidermis. To avoid too great drying of the skin it is well to anoint the body with an oil or fat after either the hot or cold pack.

Very hot water, as hot as can be borne, is a very effective antipruritic in certain intolerable forms of pruritus, like pruritus ani and vulvæ.

For this purpose the water, as hot as can be borne, should be applied on compresses which are changed every few seconds until the itching is relieved.

Wet Dressings.—The constant application of watery solutions to the skin is made in the form of wet dressings. These not only bring the remedial agents in direct contact with the surface, but also afford protection and drainage, and are more or less cooling and therefore soothing to irritated surfaces. Wet dressings may be applied either under an impervious covering or without such a covering. When it is wished to obtain the greatest amount of soothing effect from wet dressings they should not have an impervious covering, in order that the dressing may remain cool from evaporation from the surface. Under other circumstances, as, for example, when it is desired to get the effect of heat and moisture (a poultice effect) wet dressings should be covered with an impervious outside layer, such as paraffin paper or thin rubber tissue. If, as is usually the case, it is desired to promote free drainage, wet dressings should be applied well wrung out in order that their absorptive power may be greatest, and a covering applied to prevent drying. Almost all of the lotions may be used in the form of wet dressings. As a soothing, antiseptic, and slightly astringent wet dressing, boric-acid solution is one of the most useful. Normal salt solution may be used in the same way. When a stronger antiseptic wet dressing is desired, bichloride solution in the strength of 1:5,000 or 1:10,000 is our most useful application.

Lotions.—Lotions are liquid mixtures, usually with water as the menstruum, occasionally with alcohol or some other liquid of similar consistence. Plain water is ordinarily used, but occasionally for this is substituted limewater, rose water, or elder-flower water. Use is made of limewater because of its soothing, slightly astringent properties. Lotions in intensity of action may be said in a general way to lie between powders and ointments. They are ordinarily more effective applications than powders and, unless continuously applied, are less active in their effects than ointments. They furnish an agreeable and convenient method for intermittent application. They are convenient for use where an extensive surface is to be treated, or about the face or in the deep creases of the body where the use of greasy applications is disagreeable. As already pointed out, lotions are soothing through the reduction of temperature produced by their evaporation. Aside from this their therapeutic properties depend upon their composition. The intermittent application of lotions, particularly if they contain powders in suspension, is apt to cause extreme dryness of the surface. This can be avoided by adding five to fifteen drops of glycerine per ounce. Some of the lotions which are intended for intermittent application may be given a little more body and rendered demulcent by the addition, together glycerine, of tragacanth, from one to two or three grains to the ou Where it is wished, on the other hand, to increase the drying effec lotions and the rapidity of evaporation a little alcohol is added, f ten to sixty drops to the ounce. Upon an unbroken skin alcohol ma added in greater strength for its cooling effect.

Lotions are usually applied intermittently by dabbing them upon surface for a few minutes at a time. If a more positive effect is desi cloths wet with the lotion are intermittently applied. When the grea effect is desired, wet dressings wrung out of the lotion are continuo applied.

According to their properties lotions are soothing, antiprur stimulating, and antiseptic. Several of these properties are usually c bined in one preparation. The antipruritic lotions are nearly all so ing lotions, as are the astringent and stimulating lotions in their we strengths. Only nonirritating lotions are suitable for continuous ap cation to the surface.

Of the innumerable combinations which may be made in lotions, following formulæ may be regarded as among useful or as typ examples:

Boric-acid solution, either saturated (four per cent) or with e quantity of distilled water. This is soothing, slightly astringent, antipruritic. It combats the formation of pus, and applied continuo as a wet dressing is one of our most useful applications in acute infl matory diseases of the skin.

Normal salt solution may be used in the same way as boric-acid s tion, especially as a perfectly nonirritating dressing.

Solution of Aluminum Acetate (*Liquor Burrowii*).—This is eight-per-cent solution of aluminum acetate in water. Diluted with f four to eight parts water it is an extremely soothing and slightly tringent lotion, very useful in acute inflammatory dermatoses. It is chiefly in the form of wet dressings. Another good formula is:

℞ Aluminum acetate	1 to 3	per cent;
Boric acid	2 to 4	"
Resorcin	1 to 2	"
Water ad	100	"

Lead and Opium Wash.

℞ Sol. subacetate of lead, Glycerine, and Tinct. opii,	āā	♏v–xv;
Water or fresh milk		℥i.

A soothing astringent application for acute inflammatory dermat

The dilute solutions of subacetate of lead and of subacetate of lead and tincture of opium are extremely useful in acute itching dermatoses, and are widely used. Some caution should be observed in the use of solution of subacetate of lead upon the face. Subacetate of lead solution mixed with water gives a white insoluble precipitate, and if it is used upon ulcerated surfaces involving the corium the precipitation of lead salts may form a permanent whitish stain when the surface heals. This, however, does not interfere with the use of this preparation in weeping eczemas where there is no destruction of the corium. A solution of subacetate of lead in the strength of one half dram to one dram to the ounce of water, is an antipruritic lotion somewhat irritating to acutely inflamed surfaces, but valuable to subdue itching where the inflammatory process is not too acute. The mixture of lead and opium in the strength of five to fifteen minims of each to the ounce is best used in the form of wet dressings, in the same way as boric-acid solution and solution of aluminum acetate.

Similar preparations to the above for use on circumscribed surfaces are:

℞ Lead acetate,	āā	gr. i;
Morphine acetate,		
Distilled water	ad	℥i.

or,

℞ Extr. opii		gr. ij;
Liq. plumbi subacetat. dil.,	āā	℥ss.
Aqua,		

It is very common, in order to get the effect of powders upon the surface, to add to soothing and antipruritic lotions insoluble powders, which are left upon the surface after the evaporation of the water. The powders usually added are zinc oxide, calamine, prepared chalk, or bismuth. The most commonly used lotion of this sort is calamine lotion of about the following formula:

℞ Oxide of zinc,	āā	℥ss;
Calamine,		
Glycerine,	āā	♏i–v;
Carbolic acid,		
Water, or limewater	ad	℥i.

Antipruritic Lotions.

℞ Borax or sodium bicarbonate		gr. xv;
Glycerine		♏x;
Water	ad	℥i.

℞	Sol. subacet. lead	♏xv–xxx;
	Dist. water ad	℥i.

℞	Benzoic acid	gr. xv;
	Glycerine	ʒi;
	Water ad	℥i.

For pruritus and urticaria.

℞	Comp. tinct. coal tar	♏v–xx;
	Water ad	℥i.

For itching dermatoses.

℞	Carbolic acid, Glycerine, āā	♏v–xxx;
	Water ad	℥i.

℞	Alcohol	ʒij;
	Water ad	℥i.

Antipruritic evaporating lotion for unbroken surfaces.

℞	Menthol	1 to 2	per cent;
	Camphor chloral	1 to 2	"
	Carbolic acid	1 to 2	"
	Alcohol, or alcohol and water, or alcohol and distil. extr. hamamelis...ad	100	"

For pruritus in noninflammatory surfaces.

℞	Alum	gr. x;
	Water	℥i.

Astringent and sedative.

Distilled extract hamamelis.
Slightly astringent and soothing.

Lotio Nigra, or,

℞	Lotio nigra, Aq. calcis, āā	℥ss.

Soothing and astringent.

℞	Tannic acid	gr. x;
	Water	℥i.

Soothing and astringent.

℞ Alum	gr. v;
Zinc sulphate	gr. ij;
Glycerine	♏xv;
Rose water	℥i.

Astringent.

℞ Zinc sulphate, Potassium sulphuret, } āā	gr. v–xv;
Water	℥i.

Astringent and stimulating.

℞ Silver nitrate	gr. ij–x;
Water	℥i.

Antipruritic, astringent, antiseptic.

Liniments and Oils.—Liniments are liquid oily preparations. They may be made either with oil as the menstruum or with mixtures of oil and water. The oils ordinarily used are olive oil, linseed oil, liquid vaseline, and occasionally cod-liver oil, oil of sweet almond, cotton-seed oil, and other oils. The liniments which are mixtures of water and oil approximate in their properties the lotions and are used in much the same way. The liniments made of oil alone may be regarded as liquid salves. They do not protect the surface as thoroughly as salves, but they spread readily and are more easily used over very large surfaces. One of the commonest uses for oil is for removing crusts; for this purpose dressings wet with the oil are applied until the crusts are thoroughly macerated and can easily be wiped off. A favorite mixture for such use is

℞ Boric acid	ʒss;
Olive oil	ad ℥i.

The oil pack, similar to the wet pack, is a convenient and very useful measure occasionally in extensive inflammatory conditions. Cloths are dipped in a liniment or oil, such as the boric-acid and olive-oil mixture above, and the patient is then wrapped in these. Such a permanent dressing is an effective and agreeable application when large surfaces have to be covered.

Examples of liniments commonly used are the following:

℞ Calamine, Zinc oxide, } āā	ʒss;
Limewater, Olive oil, } āā	℥ss.

Calamine liniment. One dram of lanolin may be added to this to make it more of a creamy application (Crocker).

Carbolic acid (one half to one per cent) is commonly added, and any other drug soluble in water may be added.

Calamine liniment is similar to calamine lotion, and is a soothing, protective application.

℞ Limewater	℥ss;
Linseed or olive oil	℥ss.

The well-known carron oil, much used formerly for burns; soothing, but mussy and disagreeable about the face or on hairy surfaces.

℞ Lard, Limewater, } āā	℥ss.

When this is shaken up it forms a temporary thick emulsion which is a bland, cooling liniment. Carbolic acid, boric acid, and other drugs may be added to it.

℞ Carbolic acid	5	per cent;
Olive oil	95	"

Stimulating and antipruritic liniment.

℞ Carbolic acid	ʒi–ij;
Liquor potassæ	ʒi;
Linseed oil	℥i.
Oil of bergamot	q. s.

Shake before using.

Bronson's antipruritic oil. A stimulating antipruritic application.

℞ Turpentine	ʒi;
Oil of cade	ʒi;
Olive oil	ad ℥i.

Stimulating liniment for psoriasis (Crocker).

Ointments.[1]—Ointments are solid or semisolid preparations of fats or fatlike substances with which may be incorporated various other remedies. They are the most commonly used of external applications.

The functions they serve are various. They supply fat to the skin; they afford a vehicle for the application of remedies; they furnish a protective covering to the skin, especially against air and water; they hinder evaporation from the surface and prevent the drying of the skin; and they are to a certain extent detergent, softening crusts and scales upon the surface and rendering their removal easy.

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[1] Ointments and pastes: E. Wende, *Amer. Medical Quarterly*, June, 1899. Preparation and use of ointments: Bulkley, *Therapeutic Gazette*, August 15, 1891.

Ointments should possess the following properties: They should be smooth and homogeneous and spread evenly. Their melting point should be somewhat above the body temperature, so that when applied to the surface they soften but do not too readily liquefy. They should be non-irritating except when drugs are added to give them that property. They should be free from grittiness, and to that end drugs incorporated with them should be in solution or in impalpable powders. They should be free from rancidity; if possible, therefore, they should be physically and chemically stable, and, if they are not, they should be used only when freshly prepared or should be kept at a low temperature, not above 59° F., so that decomposition will not readily occur.

The defects which are most frequent in ointments are rancidity and the presence of gritty particles of the drugs incorporated with them. Rancidity arises from the decomposition of the fats into their fatty acids and glycerine, both of which are irritating, and is very apt to occur unless carefully guarded against. Palpable particles in ointments arise only from careless preparation, but are of common occurrence.

The fats which are ordinarily used in the preparation of ointment bases are lard, lanolin, vaseline, olive oil, oil of sweet almonds, liquid vaseline, paraffin, white and yellow wax, cocoa butter, spermaceti. The combinations of these are innumerable, the different fats being mixed especially with the view of giving proper consistence to the ointment base. Hard fats, like spermaceti, cocoa butter, wax, and paraffin, are added to give stiffness; glycerine, oil of sweet almonds, olive oil, liquid vaseline, to soften.

As to the relative absorbability of fats, there is a good deal of diversity of opinion. Lanolin being a fat analogous to skin fat, is usually said to be taken up most readily by the skin; similar strong claims are made for certain other proprietary preparations, like vasogen. The accuracy of these claims is open to question. For example, Aubert[1] found that oils and lard penetrated best with simple unction, but if friction was used lanolin was most penetrating. Luff,[2] on the other hand, found that vaseline preparations were most rapidly absorbed and lanolin most slowly. According to my clinical experience there is little practical difference in the relative absorbability of different ointments. Any of them will be absorbed upon sufficient friction, and if absorption is desired, the most important point is to have the ointment of proper consistence for easy friction without having it too soft.

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[1] *Journal of Cutaneous and Genito-Urinary Diseases*, December, 1892.

[2] *Monat. f. Prak. Derm.*, Bd. XI, Heft 2.

The ointment bases which are ordinarily used are benzoinated lard, cold cream, vaseline, and lanolin. Others less frequently used are Hebra's diachylon ointment, Unna's casein ointment, resorbin, mollin, epidermin, and other special formulæ. Benzoinated lard, cold cream, and vaseline are all rather soft, and for use in warm weather or when an ointment of greater firmness is needed it is well to add from five to twenty per cent of white or yellow wax or paraffin. Benzoinated lard, vaseline, and lanolin are all occasionally irritating. This is seldom if ever true of cold cream.

Benzoinated lard is the most frequently used ointment base. Its chief objection is that it is very liable to become rancid, and for this reason it should not be made the base of ointments intended for application on acutely inflamed surfaces unless known to be fresh. Its melting point is 100° to 104° F., and it is too soft to make a firm ointment.

A thicker ointment base analogous to benzoinated lard is simple ointment, the unguentum of the United States Pharmacopœia, which consists of eighty per cent lard and twenty per cent white wax.

Vaseline or petrolatum is not a fat in the chemical sense, that is, it is not a combination of glycerine with fatty acids, but is a mixture of hydrocarbons. Its physical properties are, however, those of a fat. Vaseline has the great advantage of stability and does not become rancid. It has the disadvantage occasionally of being irritating to an individual. In prescribing it care has to be used to obtain a reliable product free from irritating properties.

Lanolin, or adeps lanæ hydrosus, and similar preparations are purified wool fats. It is especially claimed for them that they have greater absorbability than other fats, a claim which is not established. Lanolin has the advantage of being freely miscible with water. An objection to it is that it is too sticky for use alone as an ointment, and cannot readily be spread upon a surface. In the proportion of twenty to fifty per cent, however, it is a good addition to ointments for the purpose of rendering them firmer and more adhesive; when mixed with an equal quantity of rose ointment or vaseline, or with thirty per cent of olive or almond oil or liquid vaseline, it makes a smooth ointment of proper consistence.

Cold cream, unguentum aquæ rosæ, a combination of spermaceti, white wax, almond oil, and rose water, is for most purposes the most elegant of ointment bases. It is cooling and bland and is not, like lard and vaseline, occasionally irritating to a skin. Rose ointment also does not become rancid nearly so readily as lard, but in time may become so.

Many formulæ are used which are variations upon the official formula of rose ointment.

Rose ointment contains nearly twenty per cent of water, and it is a type of a large class of bland ointments containing water which owe their cooling property chiefly, according to Unna, to the evaporation of the water which they contain.

Unna's Cooling Salve.—Unna has taken advantage of this principle to construct formulæ for cooling salves. He has suggested two forms of these, one a salve of ordinary consistence (unguentum refrigerans), the other a soft creamy salve (cremor refrigerans). The cooling salves are composed of

℞ Lanolin	10	parts;
Benzoinated lard, or a similar ointment	20	"
Water or some watery solution	30	"

The creams contain the same proportion of lanolin and benzoinated lard, but twice the quantity of water. Examples of his formulæ are the following:

℞ Lanolin	10	parts;
Benzoinated lard	20	"
Rose water	30	"

The usual unguentum refrigerans.[1]

℞ Lanolin	10	parts;
Ointment of zinc oxide	20	"
Rose water	30	"

Unguentum refrigerans zinci.

℞ Lanolin	10	parts;
Benzoinated lard	20	"
Distilled water	24	"
Ichthyol	6	"

℞ Lanolin	10	parts;
Benzoinated lard	20	"
Rose water	60	"

The usual cremor refrigerans. It may be made with limewater.

℞ Lanolin	10	parts;
Vaseline	20	"
Sol. peroxide of hydrogen	20–40	"

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[1] Duhring published this formula for a "soft creamy ointment" in 1895 ("Cutaneous Medicine," p. 205).

Engman's Salves.—Engman has recently suggested salves and creams:

CREMOR LANOLINI (*Engman*).

℞	Oil sweet almonds	8 o
	Lanolin	8 o
	Limewater	1 p
	Oil of rose	q. s.

Melt the lanolin and oil of almonds together, and add t and oil of rose while cooking, stirring constantly. Keep in

UNGUENTUM CREMOR (*Engman*).

℞	White wax, Spermaceti, āā	1 oun
	Lanolin	4 oun
	Oil of cocoa	1 oun
	Oil of sweet almonds	8 oun
	Tr. benzoin	30 drop
	Orange flower water	2 ounc

Melt the oil and wax together and add the Tr. benzoin and flower water while cooling, stirring constantly. Keep in ice box.

CREMOR TRAGALANI (*Engman*).

℞	Tragacanth	3 drams;
	Water, hot	8 ounces.

Make paste and add cremor lanolini 8 ounces.

Diachylon Ointment.—Diachylon ointment is an oleate of lead ointment which was suggested by Hebra. It is a valuable astringent soothing ointment alone, and is also an excellent ointment base. When properly made it is a smooth yellowish ointment of the consistence of butter. Unfortunately it is difficult of preparation, and is apt to become rancid. Various formulæ have been suggested for its preparation. The best preparation is made as follows (Hyde):

Fourteen ounces of olive oil and sixteen of water are mixed and brought to the boiling point over a water bath. Thirty drams of finely powdered litharge are slowly sifted into the liquid, which is boiled, with constant stirring, until all the particles of litharge have dissolved and a homogeneous mass is formed. Water is added as necessary during the boiling to preserve the proper consistence, and stirring is continued until the ointment is cold. While cooling, one drop of oil of roses or oil of lavender is added to each two ounces of ointment.

A simpler formula, and one which produces a satisfactory smooth but slightly stiffer ointment, is that suggested by Piffard of melting together equal parts of lead plaster and vaseline. This has the great advantage of not decomposing.

Vasogen is a mineral fat similar to vaseline, which has been recommended as preferable to vaseline because of more ready absorption by the skin. Like vaseline it is obtainable in both the liquid and semisolid forms. It is offered commercially in some eligible mixtures, especially the vasogen mercury ointment, but I do not believe that it possesses any essential advantages over vaseline.

Mollin is a soft soap with which about seventeen per cent of free fat is incorporated. It possesses the advantage of being miscible with water and being easily removed by water, and it is a good vehicle for most drugs. It cannot be used upon acutely inflamed surfaces.

Naftalan is a dark-brown neutral mineral fat similar to vaseline but of harder consistence. In its therapeutic properties it resembles tar. It is less active, however, and is not nearly so irritating to acutely inflamed surfaces. It is offered as an ointment base, but is rather to be regarded as an active remedy which, on account of the coal tar which it contains, may be used as a substitute for tar.

There are several special ointment bases which have the consistence of a thick cream and are really between ointments and liniments. The chief of these are the following:

Unguentum Caseini (*Unna*).

℞ Casein	14	per cent;
Alkalies	0.43	"
Glycerine	7	"
Vaseline	21	"
Water	ad 100	"

This forms a uniform thick, viscous, whitish emulsion. Rubbed on the skin it dries into a smooth elastic coating which does not exert any tension and requires no previous heating. It is contraindicated for hairy surfaces. Calcium salts and all acids rob this preparation of its property of forming a solid covering. Slightly acid substances, such as tar and balsams, may be mixed with it to the extent of twenty per cent, but it is well to add at the same time green soap in the proportion of one part to four of tar. Neutral substances and powders may be combined with this preparation to the extent of twenty per cent, provided the same amount of vaseline is added. It is applied by rubbing the salve upon the affected area with the moistened hand.

Resorbin (*Lederman*) is a fat emulsion consisting of almond wax, water, a small amount of gelatine, and a slight addition of la lin. It disappears quickly on a little massage, leaving behind onl slight amount of fat. On account of its water contents it acts as a frigerant and antiphlogistic. It is highly recommended as a base mercurial ointment.

Epidermin (*v. Hebra*) is a rapidly drying salve of milky half-l consistence, consisting of beeswax, water, and glycerine. It is po does not exert pressure, and is fairly permanent.

Unguentum Domesticum (*Unna*) consists of yolk of egg (1 per cent), almond oil (fifty-nine per cent), and balsam of (one per cent), and is useful on account of its emulsion-for properties as a medium for tar, ichthyol, and substances of si consistence, bringing them into the form of salves and formi smooth, shining, rapidly drying covering. On account of sulphur contents of the egg yolk it acts also as a mild sul salve. It is especially suited for the therapy of eczemas, acne, scabies.

Pastes.—The term paste is applied in dermatology to fatty a cations in which there is incorporated with the ointment base a larg portion of powder. The effect of this is to give them more bod render them stiffer and more adhesive than ointments, so that they nish greater protection. Further than this, their chief advanta that from the large proportion of powder in them they have a con able capacity for absorption of secretions, so that secretions are not to accumulate under them to the same extent that they do under ments. They may be used therefore to better advantage than oint upon weeping surfaces.

The proper method of applying pastes is to rub them upon the in a thick, smooth layer, which should then be dusted over with an powder, or, better still, covered with a layer of gauze or of abso cotton. They are easily removed by the application of oil or o vaseline.

Lassar's Paste.—The type of all the pastes is Lassar's paste.

℞ Zinc oxide, } āā .... ʒij;
Starch,
Vaseline .... ʒiv.

As originally suggested, this contained two per cent salicylic acid, may be either added or omitted, as indicated. Manifestly there ar drugs which cannot be incorporated with this paste.

Other pastes which may be given as examples of these applica are as follows:

Ihle's Paste.

℞ Zinc oxide, Starch, Lanolin, Vaseline, āā	ʒij;
Resorcin	gr. x.

℞ Boric acid	ʒi;
Zinc oxide, Starch, āā	ʒiij;
Vaseline	ʒxij.

A softer paste than Lassar's. "Boric acid possesses in a notable degree the property of stiffening pastes and ointment" (Duhring).

℞ Lanolin	65 parts;
Paraffin	30 "
White wax	5 "
Mix and add Water	30 "

Very stiff, adherent, quickly drying paste (Duhring).

Unna's Paste.

℞ Terra silicea	ʒi–ij;
Zinc ointment	ad ℥i.

Plasters.—Plasters are adhesive applications which are chiefly employed where one wants to apply over a small area very active remedies. They have as a base mixtures of wax, resin, and lead plaster. Their chief value is for the application of caustics, and they are not very largely used in dermatology. Examples of them are the official soap plaster and the following:

℞ Burgundy pitch	ʒx;
Resin, Yellow wax, āā	ʒiv;
Oil of turpentine	ʒij;
Canada balsam	ʒss.

A closely adherent plaster for use with pyrogallic acid, resorcin, and other caustics (Duhring).

Neumann's Plaster.

℞ Oil of turpentine	1 part;
Yellow wax	1½ parts;
Lead plaster	6 "

Salve Mulls and Plaster Mulls.—As a substitut forms of fatty applications Unna has suggested salve mu mulls, which are furnished with the ointment or paste upon cloth. The salve mulls are ointments or pastes sp the plaster mulls are similar, but are incorporated in a which makes them adhesive. These mulls are furnished in various formulæ. They are a convenient form of applica surfaces, but a vital objection to their use is that one mus formulæ. A further objection is that it is impossible a manufacturer—and they are made in Germany—to obtain in sufficient variety to be practically available. They offer advantages over less expensive and more adaptable applicati

Pencils.—Unna has introduced, as a substitute for pastes for application upon small surfaces, salve and paste pencils. sist of various medicaments incorporated with fat mixtures hard enough to mold into pencil form. The salve pencils are wax, oil, and resin; the paste pencils with starch, dextrin, su tragacanth. Various medicaments, like salicylic acid, arsenic, sive sublimate, cocaine, ichthyol, iodoform, chrysarobin, are in rated with them. Manifestly the field of their application is limited.

Fixed Protective Applications.—Under this head I include d ings which are applied in liquid form to the surface and upon dr leave a solid adhesive layer. These are for certain purposes exceedi useful and convenient forms of applications. In the first place, t do not rub off after they become dry, and they are for that reason c venient. Their chief function is to apply a firm protective covering the surface, which not only protects against the air and other exter chemical irritants, but is firm enough to furnish considerable prot tion against scratching and other forms of traumatism. They also f nish a fairly satisfactory vehicle for the application of various act remedies, but remedies which are added to them should usually be so what stronger than would be allowable were they in ointments or lotic

These fixed protective applications may be divided into two ki as regards their solubility in water: the insoluble dressings and soluble dressings. The insoluble dressings have in solution substan which are not soluble in water and which, therefore, when dried u the surface are not affected by water or by secretions. They have alco ether, or similar solvents as the menstruum, and consequently are adaptable to lesions which cannot be temporarily irritated. Their c use is where a firm protective dressing is desired over a small surf They also occasionally serve as very convenient vehicles for the appl tion to limited areas of remedies, like chrysarobin, whose action i desired to limit or whose application in less firm vehicles has some disagreeable feature. The simplest of these fixed dressings, which in this case amounts simply to a varnish, is tincture of benzoin or compound tincture of benzoin. These, upon the evaporation of the alcohol, leave a thin layer of varnish upon the surface which under certain circumstances affords considerable protection and furnishes a good means of application of agents soluble in alcohol, like corrosive sublimate. The type of dressings of this class is collodion, which, upon the evaporation of its ether, leaves on the surface a firm, closely adherent, transparent layer of pyroxylin. This layer, while insoluble in water, does not prevent the evaporation of perspiration, so that it does not macerate the skin.

A similar preparation to collodion is liquor guttæ perchæ or traumaticin, a ten-per-cent solution of gutta percha in chloroform. This is used especially as a vehicle for chrysarobin.

Another of these dressings is filmogen, which is a solution of nitrocellulose in acetone, with the addition of a certain amount of oil.

With all of these may be incorporated inert powders and the various drugs which are soluble in them.

The soluble fixed dressings are solutions of gelatine or similar substances which are either liquid or can be liquefied by moderate heat, and which when spread upon the surface dry into an adhesive layer. As the solvent in these dressings is water, they can be applied to acutely inflamed surfaces without producing irritation. These dressings are firm enough to furnish sufficient protection against scratching and also against the irritation of the air, while they allow free evaporation of the secretions from the surface. A minor advantage is that they are easily removed with water. The most useful of these are Unna's zinc oxide jellies, the formulæ for which are as follows:

℞ Zinc oxide	15	parts;
Gelatine	15	"
Glycerine	25	"
Water	45	"

Soft jelly.

℞ Zinc oxide	10	parts;
Gelatine	30	"
Glycerine	30	"
Water	30	"

Hard jelly.

Chrysarobin or ammoniated mercury (five to ten per cent), iodoform or sulphur (fifteen to thirty per cent), can be added to either the hard or soft. Salicylic acid, resorcin, naphthol, creosote, carbolic acid, can be added up to ten per cent to the hard. Tar and ichthyol may be incorporated with the hard form up to thirty-three per cent.

Unna's jelly when cold is firm and cannot be spread. To apply it, it is heated over a water bath until it liquefies, and is then painted on the surface with a brush. It forms a thin sticky layer at first, which in the course of about an hour dries into a flexible smooth layer that is comparable in its smoothness and flexibility to a thin kid glove. The dressing is made more durable if a layer of gauze is spread over it while it is still moist, and if the dressing is powdered with talcum after it dries. The same purpose is accomplished by covering the dressing while still soft with absorbent cotton and removing the superfluous cotton after the jelly dries.

Unna's jelly cannot be used satisfactorily on surfaces which ar weeping profusely, as the secretions dissolve it off. It is chiefly serviceable for dry localized forms of inflammatory dermatoses.

Other forms of jellylike application are made from tragacanth. Th type of these is Pick's linimentum exsiccans, a modified formula fo which is:

℞ Tragacanth	5	parts;
Glycerine	5	"
Boiling water	6	"
Boric acid	4	"

This forms a smooth, soft jelly which can be spread upon the surfac and readily dries, leaving a thin protective covering. It is a coolin soothing application, and is an agreeable preparation for use in sul acute eczemas on the face and hands. It is rendered firmer by the add tion of three to four per cent of oxide of zinc, and other inert powde can be mixed with it. Resorcin, salicylic acid, carbolic acid, and oth drugs may be incorporated with it.

This formula of Pick's gives a jelly which decomposes upon keepin The addition of one half per cent carbolic acid preserves it.

A very useful preparation which is similar to Pick's jelly, but muc thinner, is made with the following formula:

℞ Tragacanth	1 to 2	per cent;
Glycerine	4	"
Boric acid	4	"
Water ad	100	"

This is made more agreeable by the addition of

Oil of rose	♏viij;
Oil of lavender	♏xx;
And oil of bergamot	♏xl to the gallo

It forms a smooth cream lotion which is very soothing.

Other preparations similar to Pick's jelly are Elliot's bassorin paste and gelanthum recently suggested by Unna, a mixture of which the base is gelatine and tragacanth.

LUSK'S ANTISEPTIC PAINT.[1]

℞ Acetanilid	ʒi;
Zinc oxide	ʒiij;
Iodized starch, 5 per cent....................	ʒiv.

The starch is to be freshly prepared and the acetanilid to be finely powdered. When the mixture is used, add sufficient water or liquid vaseline to make a paint. It turns white on contact with pus. It is an excellent application where an antiseptic is needed.

Soaps.—Soaps are of two kinds, hard or soda soaps and soft or potash soaps. The chief use of soaps in dermatology is for their detergent action, and the most useful soap to the dermatologist is green or soft soap (sapo mollis, sapo viridis), a potash soap containing from one to four per cent of free caustic potash. This has a large field of usefulness as a detergent where a free alkali is indicated, both to dissolve off fats and to loosen horny epithelium. It is also used where it is desired to soften the superficial layer of the horny epidermis and at the same time stimulate the skin. For many purposes green soap is conveniently used in the form of tincture of green soap (linimentum saponis mollis, spiritus saponis kalinus), a mixture consisting approximately of two parts of soap and one part of alcohol.

Mollin has already been referred to.

To avoid the ill effects of soap upon acutely inflamed surfaces, where their use is to a certain extent necessary for cleanliness, we are often hard put to find a soap free from alkalies and as slightly irritating as possible. For this purpose Unna has introduced superfatted soaps which contain an excess of fat rather than of alkali. The unmedicated superfatted soap of Unna, the "basic" soap, is a very bland soap, but is not better than the well-made neutral toilet soaps.

Many medicated soaps are upon the market with which are incorporated corrosive sublimate, tar, sulphur, salicylic acid, resorcin, carbolic acid, and various other drugs. They have a restricted field of usefulness. The amount of drugs which is applied in this way is very minute, but has some effect; for example, a corrosive sublimate soap or a soap of salicylic acid, resorcin, and sulphur, if used two or three times a day, will produce some exfoliation of the skin and some irritation, such as would be expected from the application of these drugs. Any considerable therapeutic effect, however, from the drugs incorporated in these soaps is impossible, and their action is at best uncertain. According to the investigation of Curzio, quoted by Crocker, drugs vary greatly in their action when incorporated in soaps. One per cent sublimate soft soap was neither aseptic nor antiseptic, even after twenty-four hours' contact; sublimate hard soap after twenty-four hours produced some effect; ten-per-cent carbolic acid soap had no antiseptic effect; while salicylic acid (three per cent) and boric acid (five per cent) are both antiseptic.

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[1] *Journal of Cutaneous Diseases*, December, 1901.

Mechanical and Physical Agents

Heat.—Heat applied in the form of hot water constringes the blood vessels and is distinctly sedative. It is a very useful application in inflammatory processes involving the connective tissue. It is valuable to relieve itching and subdue irritation generally in acute inflammatory dermatoses, like acute eczema. For these purposes hot water is applied in the form of compresses for a few minutes at a time at frequent intervals. As a means of relieving itching in certain intolerable forms of itching dermatoses where the inflammatory process is not very acute it is a very useful agent, to which reference has already been made under the use of water.

Dry, radiant heat, such as can be gotten from a powerful incandescent electric light, is at times effective in the relief of itching and pain in the skin.

The use of heat for its destructive effect in the form of the thermocautery needs only to be mentioned.

Massage.—The use of massage for its constitutional effects in diseases of the skin is controlled by general indications. Gentle general massage given in the evening is often soothing to nervous patients, and tends to relieve itching. When patients are unable to take exercise in chronic diseases, massage may be used as the best available substitute. In dermatoses associated with chronic constipation due to intestinal atony abdominal massage is often of signal benefit.

The use of massage for its local effect upon the skin has been extensively tried, but its value has not proved great. It is used with benefit to stimulate the scalp in premature and nervous alopecia. To stimulate the sluggish skin in acne it is of considerable benefit. In scleroderma and elephantiasis, and in Raynaud's disease and other circulatory disturbances, the stimulation of the circulation which massage causes is grateful and may be of some benefit. Among irregular dermatologists massage is used very largely, and the most exaggerated claims are made for it. Massage of the face where the skin is sluggish temporarily improves its color and, when used with bland ointments, cleanses it, and is thus a cosmetic of some use. Beyond this its usefulness as a cosmetic agent is small. There is little reason to believe that it ever removes wrinkles or undesirable accumulations of fat, like a double chin—purposes for which it is used most assiduously.

Electricity.—Both general galvanism and faradism exert a certain amount of tonic as well as soothing effect which can be made use of in the treatment of certain dermatoses, particularly those itching dermatoses which are dependent upon nervous disturbances. In such cases electricity is applied, as under other circumstances, for its constitutional effects.

High Frequency Currents.—In the last few years very elaborate attempts have been made to apply the currents of very high potential and rapid alternations which are produced by the so-called high-frequency apparatus. For use in skin diseases high frequency currents are applied by means of their effluvia, or brush discharges. The ordinary method is to attach one terminal from the high frequency apparatus—usually a resonator—to an electrode, which consists of a glass vacuum bulb. When this electrode is brought near the skin there passes a violet brush discharge, which is accompanied by some tingling if the electrode and the skin are not in contact, but with almost no sensation when they are in contact. The result of the application for several minutes of these "effluvia" is a hyperæmia which persists from a few hours to a day. The effect of high frequency currents is that of stimulation, and they are accordingly recommended in various inflammatory dermatoses, like eczema, psoriasis, lupus erythematosus. They are also recommended for the destruction of pathological tissues, like rodent ulcers and lupus. The high frequency applications are further said to be sedative, and are recommended for the relief of pruritus. In my experience their use has been very disappointing, and I am not convinced that they are of any more value than are the ordinary brush discharges from a static machine. They at least have the advantage of being not uncomfortable.

Electrolysis.—Electrolysis, as the term is used in medicine, refers to the decomposition of tissues by a galvanic current. When needles connected with a galvanic current are introduced into living tissues, there is destruction of tissues around both needles with the production of acids (oxidizing agents) at the positive pole and alkalies (reducing agents) at the negative pole. The value of electrolysis in dermatology, therefore, lies in the fact that it gives us an easily controllable method for the destruction of minute masses of tissue. We avail ourselves of this in many procedures. For example, electrolysis offers the only practical method of destroying a hair follicle and thus preventing the growth of its hair. In the same way it can be used to destroy the channels of blood vessels in the skin; and, of course, it can be used for grosser destructive procedures, like destruction of warts and moles. One lesser advantage of its use is that the method is sterile in itself, the chemicals produced at the poles being active bactericides. In electrolysis destruction of tissue is more rapid and coagulation of albumen and the formation of a blood clot is more active around the negative pole than around the positive, and on that account it is preferable to attach the needle to the negative pole for most purposes. Another reason for using the negative pole, if needles of steel or other oxidizable metal are used, is that oxygen is given off at the positive pole, and if the needle is connected at this pole a deposit of the metallic oxide will take place in the tissues, forming a permanent stain.

For electrolysis there are necessary a galvanic battery, electrodes, and needles. The most convenient battery is the ordinary battery of bichromate cells; five to ten cells are sufficient. The current which should be passed through the tissue varies from two to ten milliampères; for removing hairs or the destruction of small lesions of the skin one to three milliampères are sufficient; for the destruction of larger lesions stronger currents may be conveniently used, eight to ten milliampères, but such currents are distinctly painful. The usual electrode is a sponge electrode on a wooden handle. With a little use it becomes rather dirty, and a much better electrode is one in which the sponge is replaced by a metal disk, say one and a half inches in diameter, which can be covered as often as desired with fresh gauze. Before using the electrode it must be dipped in water, to which it is a good plan to add a few grains of common salt; distilled water is not satisfactory because it is not a good conductor. The electrode is usually held in the patient's hand; if one's battery is weak, it is a good plan in order to diminish the resistance of the tissues to have the patient hold the electrode close to the point to be treated. A needle holder is necessary, and the usual forms obtainable from instrument dealers are satisfactory. For needles the ordinary steel jeweler's broaches are usually recommended. These, in my opinion, should not be used. They are too large and cause too much destruction of tissue; they are stiff and unwieldy; and a greater objection to them is that if the mistake is made of attaching the needle to the positive pole a permanent spot of iron oxide is left in the skin. Iridio-platinum needles are also recommended. They are objectionable because they cannot be bent. The best needle is a needle made of platinum wire. This can be bent in any direction, so that it can be conveniently used in any situation, and it is not oxidizable. For the removal of hair the best needle is made of a piece of platinum wire one and a half inches long, of which one half inch is left the width of a small sewing needle to serve for insertion in the needle holder, and the remainder is ground down until a very fine wire is formed; the only limit to its fineness is that it must retain sufficient stiffness to be able to penetrate a hair follicle. For the destruction of dilated blood vessels in the skin, and warts and similar growths, a somewhat coarser platinum needle is better.

Cataphoresis.—When a continuous electric current is passed through two liquids separated by a porous but ordinarily impermeable membrane, a flow of the liquid and of substances in solution in it is set up through the membrane from the positive toward the negative pole (cataphoresis). The skin furnishes such a membrane, and advantage can be taken of this fact to introduce drugs in aqueous solution into the skin. To do this the positive pole is moistened with the solution. This pole is placed at the desired point, and the circuit is completed by making contact with the negative pole at any point. Cataphoresis is more prompt if the surface is first washed with ether to remove the fat.

Cataphoresis has not had very extensive practical application. The difficulty about it is that one cannot tell how much of the drug is being used. It is sometimes used for producing cocaine anæsthesia, but when so used must be done cautiously. A one-per-cent solution of cocaine is sufficiently strong with a current of five milliampères to produce almost complete anæsthesia in five to seven minutes. A five-per-cent solution will in the same time produce complete anæsthesia.

In the same way other drugs soluble in water or alcohol may be introduced through the skin. Usually the process is quite painless, but with irritating drugs, like corrosive sublimate, there may be some pain.

Fulguration.—This procedure consists of the local application of the high-frequency spark by means of insulated, pointed metallic electrodes. It is really a high-frequency caustic spark. Its action is either stimulating, or mildly or strongly escharotic, according to the strength of the current, the length of the spark, and the duration of the application. We have much more controllable escharotics and the value of the method is not yet established.

Radiotherapy [1]

In recent years an entirely new field of therapeutics—especially cutaneous therapeutics—has been opened up by the discoveries of the effects upon living tissues of highly actinic forms of radiant energy, like x-rays and the rays of light at the violet end of the solar spectrum. The pioneer in this field, as far as practical work is concerned, was Finsen in his work with the ultraviolet light rays. However, the therapeutics of the x-rays or Roentgentherapy, while covering almost the same field as the therapeutic use of light or phototherapy, is more comprehensive, and for that reason is more conveniently considered first.

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[1] It is impossible in the space available here to more than sketch in the briefest way Roentgentherapy and phototherapy. For a complete consideration of the subjects the student must be referred to special works.

Roentgentherapy.—The rational indications for Roentgentherapy rest upon the effect of x-rays upon living tissues. Briefly described, the gross manifestations of x-rays upon human tissues are first a stimulation of the formation of pigment in the skin and an erythema or superficial dermatitis, both of which are indistinguishable from the tanning and sunburn produced by sunlight. The maximum effects of x-rays, however, are more intense than the maximum effects of sunlight, and if the exposures pass the point of producing a dry red dermatitis, there occurs as the next step a bright red vesicular dermatitis, after this a cyanosis of the skin, and, if the process goes further, a necrosis of the skin which results in tough, closely adherent sloughs which show little tendency to separate from the living tissues, and are accompanied by intense pain.

The changes produced by x-rays upon living tissues and upon bacteria may be briefly summarized as follows:

(1) They cause atrophy of the appendages of the skin.

(2) They have a destructive action upon organisms in living tissues.

(3) They have a peculiar effect upon the nutrition of the living cells, producing in their less intense action a stimulation of the metabolism of the tissues, which, when their effect is greater, may go on t the point of the disorganization of the cells and their destruction.

(4) This destructive action upon living cells destroys certain diseased cells before it destroys the more resistant healthy cells of th stroma.

(5) They have an anodyne effect.

From the foregoing it may be deduced that x-rays have possible indications in the following groups of affections:

(1) Conditions where it is desired to remove hair: (*a*) hypertrichosis, (*b*) sycosis, (*c*) favus, (*d*) tinea tonsurans, (*e*) tinea barbæ or tine sycosis.

(2) Where it is desired to cause atrophy or diminution in size o functional activity of the sebaceous glands: (*a*) comedo, (*b*) acne, (*c* acne rosacea, (*d*) lupus erythematosus (?), (*e*) seborrhea.

(3) Where it is desired to cause atrophy or diminution in the functional activity of the sweat glands: (*a*) hyperidrosis.

(4) It is possible that x-rays might be of use also where one wante to cause exfoliation of the nail substance.

(5) Their destructive effect upon bacteria in tissues, of course, come into play in a number of the affections in which their use is suggeste above. Such a quality offers possibilities of the widest application i bacterial diseases of the skin. Their use in lupus vulgaris is the mos brilliant application in bacterial diseases which has yet been made. Bu in this condition, doubtless, their effect in destroying tissues of low r

sistance is of greater moment than the germicidal effect. Other bacterial diseases in which they have been used successfully, and in which this characteristic is an important factor, are sycosis, acne, various forms of tinea and favus.

(6) Their stimulating effect upon the metabolism of the skin offers a wide field of application. It is probably this effect that explains the success that has followed their use in chronic indurated eczemas, lupus erythematosus, lichen planus, psoriasis, and in fact in the entire class of indurated, inflammatory diseases of the skin in which stimulation of the tissues is necessary in order to cause absorption of inflammatory products.

(7) Their power of causing the destruction of tissues of low resistance without the destruction of the healthy stroma is the theoretical indication for their use in various malignant diseases and in other processes in which we have to do with cells of low resistance. This group includes, of course, most important affections; as carcinoma and sarcoma, tuberculosis, pseudoleukæmia, leukæmia, and mycosis fungoides.

(8) Their anodyne effect comes into play in the treatment of painful malignant and inflammatory conditions, in neuralgias, and in itching dermatoses.

It is manifest from the foregoing summary of the therapeutic indications for x-rays that their field of application in dermatology is very extensive, and, as a matter of fact, this is true. It is hardly too much to say that Roentgentherapy is the most widely useful addition to the treatment of skin diseases which has been made.

In the therapeutic application of x-rays the principle of treatment is to produce a sufficient effect for the purposes of the case without overstepping the bounds of safety. In skin diseases, except in such conditions as epithelioma and lupus, where the situation warrants some risk of burn, it is always necessary to keep the x-ray reaction well below the point of active dermatitis. All of the various technics of Roentgentherapy are directed to this end. It would far exceed the space available here to consider in detail these different methods of application. The method of application which I use and have found safe consists essentially in weak exposures frequently repeated until relief is obtained or until slight evidence of x-ray effect is produced. When this occurs the exposures are at once suspended and are not renewed for two or three weeks, or until all possibility of the appearance of x-ray effect has passed. The amount of x-rays used is just sufficient to light up the tube with a faint greenish or greenish-yellow glow. The exposure is continued for from five to fifteen minutes with the tube—not the target—at a distance of four to eight inches, always using at the beginning exposures of minimum duration and of maximum distance. Such exposures can be repeated two or three times a week. Exposures of this sort, however, cannot be given unless the amount of x-rays in the tube is small. If exposures at this distance and of this duration are given with an abundance of x-rays, burns are likely to occur.

For Roentgentherapy a static machine or an induction-coil apparatus is necessary. The work can be done with a static machine, but it is less easily controlled and is not so satisfactory. For an induction-coil apparatus the following things are essential:

(1) A source of electricity.

(2) An induction coil.

(3) An x-ray tube, lead masks, and the accessory apparatus, such as tube holder, interrupter, connecting wires, etc.

The best source of electricity is the continuous 110-volt current. A coil can be worked with an alternating current by the use of a Wehnelt interrupter, but it is not so satisfactory. If a commercial current is not obtainable, a coil can be worked upon a storage battery furnishing eight volts or more of current.

For work in skin diseases a coil with a capacity of six- to eight-inch spark is sufficient, but a twelve-inch spark coil is better if much use is to be made of it.

The tube used for cutaneous diseases should be of low penetration or moderately "soft." It is well to possess two or three tubes of different degrees of penetration, although this is not necessary if one has a tube whose vacuum can be regulated.

Some sort of mask must be used in order to protect from the x-rays such areas as do not require exposure. The simplest protection is afforded by masks made of sheet lead, which are large enough to cover surrounding parts, and in which holes are cut to correspond with the area to be exposed. The most convenient of these masks is made of lead foil $\frac{1}{100}$ of an inch thick, of which four to eight layers are used in the mask. The advantage of the foil is that it renders the mask pliable. The lead foil should be covered with some nonconducting material, the most convenient of which is paper.

Phototherapy.—Phototherapy owes its introduction to Finsen. It may be said broadly that light has effects upon living tissues similar to those of x-rays. These effects consist in brief in stimulation of the nutrition of the cells and the production of an inflammatory reaction, as seen in the familiar phenomena of tanning and sunburn. The effects of light rays upon the skin when concentrated may produce a violent reaction, but when used to the extent to which concentration is possible in the practical application of phototherapy this reaction is never sufficient to cause necrosis of healthy tissues. It is, however, sufficient to cause destruction of tissues of low resistance, either directly from the effect of the light rays, or indirectly as the result of the inflammatory reaction. It has been shown by numerous observers that light rays have a decided bactericidal effect, much more marked than that of x-rays.

These properties of light furnish the therapeutic indications for the use of phototherapy. The indications are, in a general way, much the same as those of Roentgentherapy, with the exceptions, however, that Roentgen rays have a much greater penetration than light rays, and can be used effectively over much larger areas, so that the limitations of phototherapy are much narrower than those of Roentgentherapy.

The properties of light of which use is made in therapy are possessed in greatest degree by the so-called actinic rays of the spectrum, those at and beyond the violet end. The ultraviolet rays are the most active, but unfortunately the power to penetrate tissues is greatest at the red end of the spectrum and diminishes gradually toward the violet. The ultraviolet rays, the most active, are not available for use beneath the surface, and use has to be made in the treatment of skin diseases of the visible blue and violet and of the nearest ultraviolet rays.

Phototherapy was first used by Finsen for the treatment of lupus. Its use has been extended since to the treatment of superficial epitheliomata and to certain affections where its effect in producing a simple inflammatory reaction may be called into play, such as lupus erythematosus, rosacea, and other circumscribed chronic inflammatory dermatoses, alopecia areata, and vascular nævi. The extent of its application in therapeutics is limited by the fact that it can only be applied to a small area at one time, a circle about half an inch in diameter. A vital objection to it is that the penetration of the actinic rays is very limited. When tissues are exsanguinated, bacteria may be destroyed by prolonged exposure at a depth of 1.5 mm., their growth retarded at a depth of 4 mm.

The apparatus for carrying out phototherapy is all directed first to the purpose of concentrating a large amount of light upon a small area, and second, in order to avoid heat burns, to the removal of the heat which is concentrated with the light. Sunlight was first used by Finsen as his source of light, but practically strong arc lights alone are used now. The light is concentrated through lenses made of rock crystal, which does not absorb the ultraviolet rays as glass does, and is brought lown to an area of about half an inch in diameter. As blood prevents penetration of violet rays, the tissues are exsanguinated by two quartz lates, which are either pressed down against the skin by an attendant r fastened with bands. The light is poured through these perpendicularly upon the surface. Between these quartz plates cold water is circulated, and in all of the lamps for phototherapy provision has to be made for free circulation of water to carry off the heat. Various lamps have been suggested for phototherapy. Finsen and Reyn's is the best.

The duration and frequency of exposures vary in different conditions. With superficial lesions which can be completely exsanguinated, an exposure of half an hour may be sufficient. When a deeper effect is desired, exposures of an hour or even two hours may be required. The exposure is repeated only when the reaction has subsided. This occurs in from one to two weeks. In six to twenty-four hours after the exposure there occurs an inflammatory reaction varying in degree from a simple erythema to a bullous dermatitis. This does not cause any destruction of normal connective tissue. If vesicles or bullæ develop they dry and form scales under which healthy epidermis appears with increased pigmentation that ordinarily disappears in ten days or two weeks more. There persists for six months or longer some dilatation of the capillaries of the exposed area and increased sensitiveness, as shown by the ready occurrence of erythema upon slight external irritation.

Radium.—Radium gives off rays of three sorts: Alpha rays of very slight penetration which cannot penetrate beneath the surface of the skin; Beta rays which are identical in their physical properties with cathode rays and have slight penetration; and Gamma rays which have the physical characteristics of x-rays of the highest penetration. Beta and Gamma rays produce effects upon tissue analogous to, or identical with, those produced by the x-ray, and radium has accordingly been used with greater or less success in almost all of the conditions of the skin in which x-rays have been used successfully. Its therapeutic indications are the same as those of x-rays. Its chief use has been in the treatment of epithelioma and nævi, in which results like those produced by x-ray can be obtained. It is but fair to say, however, that the advocates of the use of radium find in it many advantages over x-rays. In a considerable experience with it, I have found its action upon the skin practically identical with that of x-rays. It has the advantage over x-rays in that it can be applied in sinuses or the orifices of the body and furnishes an effective way of applying actinic energy in these cavities. Wickham has used it with benefit in nævi, not, however, with as good results, I believe, as are obtained from CO_2 snow or liquid air.

Liquid Air and Solidified Carbon Dioxide.—Refrigeration.—Various experimenters, including White, Hartzel, Trimble, and Dade, have utilized the intense cold of liquid air for producing an inflammatory reaction or for the destruction of lesions in the skin. The difficulties in obtaining and preserving liquid air have been so great as to preclude the extensive use of the agent. Because of these difficulties I suggested the use of frozen carbon dioxide—carbon dioxide snow—as a substitute for liquid air. Liquid air has the temperature of – 182° to – 190° C.; solidified carbon dioxide, – 80° C. Liquid air, therefore, is a little more than twice as cold as frozen CO_2. Both are so intensely cold, however, that they freeze tissues instantly upon contact, and their uses and therapeutic indications are the same.

Carbon dioxide is furnished in commerce in liquefied form in iron tanks under very heavy pressure. In order to collect the CO_2 snow, the tank should be placed in a slanting position with the nozzle at the lower end, so that the liquid will gravitate to the lower end of the tank, and liquid CO_2, and not gas, will be blown out when the nozzle is opened. With the tank in this position, the nozzle is covered with chamois skin or some other heavy cloth. Then when the nozzle is opened, the escaping liquid CO_2 is caught in the cloth, and its evaporation is so rapid that it freezes itself and collects in the form of snow. The snow thus collected is in the form of a white, loose mass, which must be packed and molded. A convenient method of molding it is to pack it in hard-rubber or metal tubes of a diameter of one fourth to one inch, according to the size of the area which is to be treated. The sticks of snow made by packing in the cylinders can readily be pared with a sharp knife into any desired shape. It is difficult to get equal freezing over an area above an inch square, so that in the treatment of large areas it is better to make blocks of the snow with a face not larger than one inch square, and with these freeze the contiguous areas. For treating very small lesions, the snow should be packed very firmly in small cylindrical molds. This can be done best by hammering it down with a small hammer, using a round stick or iron rod to drive it down. If it is packed firmly enough, it can be converted into a hard, translucent ice, which can be used in pencils not larger than one sixteenth of an inch in diameter and brought to a fine point for treating minute lesions.

The principle underlying the use of CO_2 snow or liquid air is, of course, the production of an inflammatory reaction in living tissues by sudden, intense freezing. Any degree of reaction can be produced, from an evanescent superficial dermatitis to complete destruction of the frozen areas, and the agent furnishes a very convenient and easily controlled method of obtaining these results. Its chief advantage lies in the fact that with it a relatively deep inflammatory process can be produced which does not produce necrosis of the frozen area, but causes an interstitial sclerosis with destruction of more highly organized and complex structures and of diseased tissues of low resistance. The degree of reaction produced by freezing with CO_2 varies with the pressure of its application and the duration of the freezing. According to the degree of pressure exerted, freezing may be produced to any depth up to one eighth or three sixteenths of an inch. The duration of the freezing, however, is the most important factor in the effect upon the froze tissues. In the therapeutic use of the method, the freezing is carrie out for from five seconds to a minute. When beginning the use of th agent, it is best to make applications of from five to ten seconds an increase their duration as experience indicates. As a rule, for treatin vascular and pigmented nævi, applications are needed of thirty or fort seconds. In treating conditions where merely an inflammatory reactio is to be produced, freezing for from five to fifteen seconds is usually sufficient. The duration of the application should be timed with a watc in order that one may accurately control the effects and obtain an experience upon which to judge further applications. The method is useful in the treatment of numerous conditions where an inflammatory reaction or destruction of lesions is indicated, as in erythematous lupu nævi, senile keratosis, and deposits of pigment in the skin.

Treatment by Hyperemia.—Biers' Treatment.—The artifici production of active hyperemia, as suggested by Biers, has some application in infectious diseases of the skin. The principle underlyin the method is to produce artificially increased hyperemia so that th natural agencies by which infection is attacked in living tissues are increased. Biers has shown that this method is of very great efficacy i combating local infectious diseases. In cases where cutaneous lesion occur on the extremities, the method is carried out by the use bandages which interfere in part with the flow of the venous bloo On the face and other parts where constriction is not possible, hyperem can be produced by cupping. Active hyperemia can be produced by th application of hot air. The duration of the hyperemia is varied according to circumstances. When produced by cupping it is continue for ten or fifteen minutes to one half or three quarters of an hou When produced by compression it is maintained for three or four hou daily. The method is recommended in various diseases of the ski either known, or suspected to be infectious, as acne, furunculosis, syc sis, lupus vulgaris, eczema, psoriasis, keloid and alopecia areata. Th method has found a very limited application in dermatology. It is mo useful in acne and furunculosis.

Instruments Used in Treatment

The instruments which the dermatologist uses are few—small knive epilating and dissecting forceps, small curettes, etc. They can be ha of every instrument maker, and do not warrant especial descriptio It is, perhaps, worth mentioning that the best knives for operatin on the skin are the knives and needles that the oculist uses in oper tions upon the cornea.

VIII.—CLASSIFICATION

The classification of diseases of the skin has been a favorite occupation of dermatologists for a century or more. It has been a sort of game of dermatological solitaire over which has been spent many an interested hour. The terms of the problem have been about these:

Given, approximately, 300 diseases of more or less uncertain pathology; arrange them in groups according to their pathological characteristics. It goes without saying that no one has been able under these conditions to produce a perfectly satisfactory solution of the problem.

The logical arrangement of diseases is of course according to their pathology, but, until the last word has been said upon the pathology of the skin, a satisfactory and final pathological classification of dermatoses cannot be made. The best that can be done in the present state of our knowledge is to group the diseases of the skin as far as possible upon the basis of their resemblances in essential features. When possible, the characteristics upon which the grouping is based are pathological; at other times the grouping is anatomical; and again it is upon the basis of the resemblance of clinical features. Upon such an elastic scheme, a classification can be made which shows the essential relationship of numerous groups of diseases, and is upon the whole fairly satisfactory.

Hebra first undertook to group dermatoses according to their pathological relations, and most subsequent classifications have followed more or less closely his primary divisions. The classification followed in this work is practically that which has been generally adopted upon the basis of Hebra's classification, dividing dermatoses into hyperæmias, inflammations, hæmorrhages, hypertrophies, atrophies, pigmentary anomalies, neuroses, and parasitic diseases. I have chosen to change the grouping somewhat, making a group of Angioneurotic Dermatoses, one of Infectious Diseases, and one of Dry Scaly Inflammatory Dermatoses. The classification used here is as follows:

Group 1.—Hyperæmias:
    (*a*) Passive Hyperæmias,
    (*b*) Active Hyperæmias.
Group 2.—Angioneurotic Dermatoses.
Group 3.—Inflammations.
Group 4.—Dry Scaly Inflammatory Dermatoses.
Group 5.—Hæmorrhages.
Group 6.—Infectious Diseases.
Group 7.—Dermatoses Due to Animal Parasites.
Group 8.—Hypertrophies.

Group 9.—Atrophies.
Group 10.—Anomalies of Pigmentation.
Group 11.—Cutaneous Neuroses.
Group 12.—New Growths:
    (*a*) Degenerative,
    (*b*) Benign,
    (*c*) Malignant.
Group 13.—Diseases of the Appendages of the
    (*a*) Diseases of the Sweat Gla
    (*b*) Diseases of the Sebaceous
    (*c*) Diseases of the Hair and
    (*d*) Diseases of the Nails.

The dermatoses in these various groups are enun
of Contents.

THE PRACTICE OF DERMATOLOGY

SECTION II

HYPERÆMIAS

SYNONYMS: *Hyperæmiæ, Congestiones.*

Hyperæmia may be *active* or *passive.*

Hyperæmia manifests itself as redness which disappears on pressure. If active, the redness is bright; if passive, it has a more or less purplish tint.

Except for logical completeness the hyperæmias are hardly entitled to be erected into a class. Passive hyperæmia, except as it furnishes a favorable condition for the development of more definite pathological processes, is of little importance; and active hyperæmia, except as it occurs in physiological flushing, is actually the first manifestation of inflammation with its essential changes.

PASSIVE CONGESTION

Passive congestion shows in terminal areas of circulation. Its appearance varies according to its degree; in its slightest intensity the skin is a faint purplish red color, cool, and moist; in its most extreme type there is complete cyanosis, and the skin is of a dark purplish color and cold. In all degrees of passive congestion there is an increased vulnerability of the skin, so that secondary lesions, such as ulcerations from slight traumatisms, are easily produced. A congested skin is especially prone to inflammatory processes. In the most extreme type of passive congestion destructive ulceration and necrosis may occur.

Passive congestion may be due to local causes, as constricting bandages. It may be due to the mechanical obstacle which gravity offers to the return flow of blood from the extremities, as illustrated especially in the passive congestion of the legs and feet, which occurs with varicose veins. It is due to a variety of internal causes, and is especially associated with heart lesions or other organic lesions that interfere with the circulation. It is not infrequently seen as a result of exposure to cold. It may follow freezing, and a certain degree of passive congestion, especially of the hands and feet, is often present in individuals whose occupations expose them to cold. A variable amount of passive congestion, indicated by coldness, moisture, and redness of the hands and feet, is not uncommon, and is often without obvious explanation for its occurrence. It may be due simply to a feeble circulation without evident cardiac or vascular change; oftener it is apparently due to some obscure disturbance of the vascular tone. The most extreme illustrations of these disturbances are Raynaud's disease and intermittent claudication.

Passive congestion is of great clinical importance because of the frequency with which it is the essential factor in the production of eczemas, ulcers, and other pathological processes.

I.—RAYNAUD'S DISEASE

The characteristic feature of Raynaud's disease is attacks of ischæmia, which are followed by passive congestion of the terminal areas of circulation—the hands and feet, ears and nose, and, to a less degree, the rest of the face. The parts become in the end permanently more or less livid and cold, and show an increased vulnerability to pathological processes, so that destructive lesions from traumatism are common, and bullæ, ulceration, and even gangrene may occur spontaneously with, in the severer cases, partial or complete destruction of the parts. There may be in the various forms of Raynaud's disease itching, tingling, burning, and pain, the intensity varying according to the degree of the process. Raynaud's disease is considered more fully under Gangrene of the Skin.

II.—INTERMITTENT CLAUDICATION[1]

Intermittent claudication, which has been tersely described as "angina pectoris of the legs," is accompanied by circulatory and nutritional changes in the feet and legs similar to those of Raynaud's disease. Gangrene of the skin of the feet may occur.

The skin lesions of intermittent claudication are readily differentiated from those of Raynaud's disease by the fact that they are confined to the feet and legs, and are accompanied by characteristic pain in the legs after walking.

Both of these conditions are the result of vascular disturbances which are usually dependent upon or at least accompanied by endarteritis.

Crocker refers to an acquired form of passive congestion which occurs as mottling in rings, several cases of which have been exhibited in London. The lesions occur on exposure to cold in the form of purplish rings about an inch in diameter with clear centers. They appear when the parts are uncovered on the legs and thighs and disappear when they become warm. The disease is seen in individuals with other evidences of disturbed peripheral circulation.

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[1] Erb, *Deutsch. Zeitschrf. f. Nervenheilkunde*, xiii, 1898.

III.—PERNIO

SYNONYMS: *Erythema pernio, Chilblains.*

This occurs as persistent erythematous patches from exposure to cold in persons of enfeebled circulation. Its commonest location is upon the heels and borders of the feet, but it also occurs upon the fingers and the ears and nose. The parts are cold, somewhat tender, and when exposed to heat are apt to burn and itch intensely. In extreme cases the parts are almost cyanotic, and vesicles, bullæ, and ulcers may form from ordinary friction.

The condition is seen in individuals of poor peripheral circulation, and it is excited by exposure to cold. Pernio occurs especially in the young and old, and frequently in the tubercular. It improves during the warm months, but severe cases may not entirely disappear throughout the year. It is a vasomotor disturbance. Apparently the vasomotor mechanism of the affected parts has been damaged by repeated exposures to cold, until it has become permanently unduly irritable and is no longer able to maintain the normal vascular tone in the parts.

Pernio may be confused with erythematous lupus. In erythematous lupus the patches are sharply circumscribed, of a brighter erythematous color, with more or less scaling, and with considerable infiltration of the skin. Lupus erythematosus is followed by superficial scarring, is not influenced by variations in the temperature, and subjective sensations are absent or very slight.

The condition is most distressing in the feet, and treatment is chiefly directed to their relief. In predisposed individuals prophylaxis is of the greatest importance. Warm woolen hose should be put on before cold weather begins and warm loose shoes worn. Care should be taken to avoid getting the feet cold. The feet should be washed daily in very hot water, rubbed briskly, and quickly dried and powdered with a bland powder.

The general health should be maintained at as high a point as possible, and, in particular, active exercise should be encouraged. As general tonics, strychnine and arsenic are valuable, and Crocker recommends nitroglycerine. Wright, who believes that defective coagulability of the blood is an essential factor of the condition, recommends to remedy this 10 to 15 grains of calcium chloride, t.i.d.

For the relief of the intense itching and burning in the exacerbations of the condition, the frequent use of soothing applications like calamine lotion with two- to three-per-cent carbolic acid, are beneficial. When the process becomes less acute from the use of bland applications, the condition is improved by stimulation, such as is gotten from the use of friction, painting the part with tincture of iodine, the application of camphorated soap liniment, the use of alcoholic lotions, like alcohol with menthol (two per cent), or spirits of bay rum, or from soaking the parts in hot salt baths. Ross recommends the galvanic current for ten minutes three times a day, and Louis Jones an electric bath of the feet for ten to fifteen minutes daily. Such a bath is given by placing one electrode from a static machine or induction coil in a foot bath, the other on the patient's body, and then passing a current not strong enough to be disagreeable.

When the skin becomes broken in chilblains the parts are to be treated, as other cases of dermatitis, with mild antiseptic wet dressings or ointments. In such cases rest and entire avoidance of exposure to cold are necessary.

THE ERYTHEMAS

The word erythema is used in dermatology as a generic term to describe any redness of the skin. It is also used when qualified by adjectives to describe certain well-defined dermatoses.

The erythemas are of two kinds:

(1) The congestive or hyperæmic erythemas.

(2) The exudative or inflammatory erythemas.

As a matter of fact, this distinction, while desirable for clinical convenience and for systematic completeness of classification, is arbitrary; the hyperæmic erythemas are but the most superficial or earliest stages of inflammation, and show the essential changes of that process. The characteristics of the erythemas are considered further on pages 208 and 209.

I.—HYPERÆMIC ERYTHEMA

Hyperæmic erythema, as distinguished from exudative erythema, occurs in the form of irregular erythematous patches or of more or less generalized macular or maculo-papular eruptions. A physiological example of it is blushing. It may occur as the result of various pathological processes which disturb the vascular tone of the vessels of the skin.

Erythema occurring as patches of superficial redness due to constitutional causes is called *erythema simplex*, and the term should be confined to this form of erythema and not be made to include, as it frequently is, the erythematous patches which are produced by external irritants and are really examples of dermatitis.[1]

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[1] The various conditions, which are traditionally described as forms of simple erythema, but which are really well-defined inflammatory processes, are considered elsewhere and are as follows:

Erythema Simplex of external origin, under Dermatitis. *Erythema Venenatum*, under Dermatitis. *Erythema Intertrigo*, under Dermatitis. *Erythema Ab Igne*, under

True **erythema simplex** occurs in ill-defined patches of varying shades of red which disappear upon pressure. They are usually transitory, although occasionally they persist for several days. The subjective sensations associated with the condition rarely amount to more than a feeling of flushing or slight warmth.

The origin of the disturbance lies in the various conditions which may affect the tone of the blood vessels of the skin. It seems likely that the vascular disturbance is always the result of a disturbance of the controlling nervous mechanism. The disturbance may be in the vasomotor centers or in the peripheral vasomotor mechanism. It may be a physiological process, as in emotional disturbances; it may be of reflex origin; it may be due to substances circulating in the blood which affect either the central or the peripheral vasomotor control; and, finally, it may be due to external causes which, by irritating the vasomotor mechanism, temporarily disturb the vascular tone. Because of their nervous instability, erythema simplex occurs most frequently in children. It is produced in most cases by the presence of poisons in the blood, usually toxins, sometimes drugs or foods or their derivatives. The condition is closely related to, if not identical with, urticaria.

Simple erythema requires no local treatment beyond a bland dusting powder. The general condition of the patient should be looked into and the underlying causes removed.

MACULAR ERYTHEMA

SYNONYM: *Roseola.*

The second form of hyperæmic erythema is macular erythema or roseola. Macular erythema occurs as a macular or maculo-papular rash, more or less generalized and of symmetrical distribution. It is customary to divide macular erythemas into idiopathic and symptomatic roseolas.

Idiopathic roseola is a term applied, for want of a better, to those macular erythemas which are not associated with any well-defined systemic diseases, although they are the cutaneous expression of constitutional disturbances.

Symptomatic roseolas are the macular erythemas which occur with well-defined diseases. The eruptions of measles, of scarlet fever, of typhoid and typhus, the prodromal eruption of smallpox, and the early macular eruption of syphilis are all illustrations of symptomatic roseolas. Similar macular erythemas occur occasionally in various bacterial

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Dermatitis. *Erythema Solare,* under Dermatitis. *Erythema Paratrimma* is the superficial redness appearing upon points exposed to pressure which precedes the formation of bedsores. *Erythema Fugax* and *Erythema Urticans* are terms applied to evanescent patches of erythema which are lesions of urticarias.

diseases, as diphtheria, all forms of sepsis, Asiatic cholera, and cerebrospinal meningitis. The symptomatic roseolas present the same objective characteristics as the so-called idiopathic roseolas.

Idiopathic Roseola.—Idiopathic macular erythemas vary greatly in appearance. They are not sharply characteristic in their appearance, like the eruptions which are produced by such definite and active causes as are at work in the exanthemata and the other infectious diseases in which an exanthem is a regular feature. Idiopathic roseola may occur in a punctiform eruption or in irregular macules of various sizes up to that of a thumb nail. While more or less widely distributed and symmetrical, they are much better marked in some locations than in others, and are not so intense and generalized as the specific exanthemata. In some cases the face, especially around the nose, is chiefly involved, and in other cases the face entirely escapes. Occasionally idiopathic roseola is well defined and as widely distributed as the eruptions of measles and scarlet fever, and closely simulates in its eruption one or the other of these diseases. The course of idiopathic roseola is not regular. The condition is usually transitory, and disappears much more quickly than the specific exanthemata. Like all other inflammatory eruptions, macular erythema is followed by more or less desquamation, which after intense reaction may be very profuse.

Some congestion of the mucous membrane of the fauces and the mouth frequently occurs in idiopathic roseola, but this is never of the intense character seen in measles and scarlet fever.

Idiopathic roseola is associated with but slight constitutional disturbances. There may be for a day or two preceding it and at its onset some feeling of malaise with a temperature of 100° or 101° F., but even this slight disturbance soon subsides.

Idiopathic roseolas are the expression of some form of intoxication.[1] They may be produced by foods, particularly those containing products of decomposition; by various drugs, like belladonna, quinine, salicylic acid, and the balsams. They may result from gastrointestinal toxins or other toxic substances elaborated in the body, from bacterial toxins as in various infectious diseases like diphtheria and erysipelas, or from toxins introduced from without, as from the use of diphtheria antitoxin. They may be caused by the injection of normal horse serum (Berg).

Diagnosis.—The chief importance attaching to idiopathic roseola is that of its diagnosis from the acute exanthemata. At times this may be difficult or impossible, and then the only safe course to pursue is to take the necessary precautions for two or three days until time makes the diagnosis clear. The diagnosis can ordinarily be made in idiopathic by the absence of the severe constitutional disturbances of the anthemata and of the other characteristic manifestations aside e eruption. Idiopathic roseola lacks always the intense catarrhal

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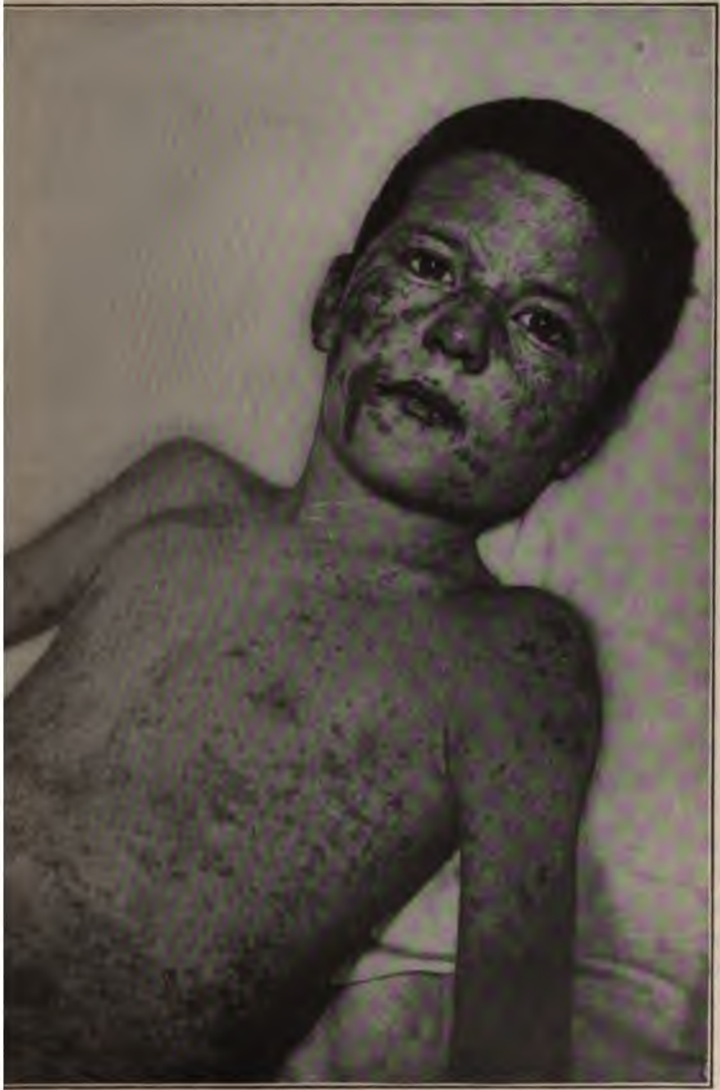
[1] Schamberg, "The Toxic Erythemata," *Jour. Cutan. Dis.*, 1904, p. 468.



29.—Rash from Diphtheria Antitoxin. (Schamberg's collection.)

s of measles and the angina of scarlet fever, as well as the pro- stemic disturbances which accompany these diseases. The con- ost likely to cause confusion is German measles (or rötheln).

Idiopathic roseola is a noncontagious disease and German measles is contagious, and a history of the contagion can usually be gotten. In German measles a characteristic feature is the enlargement of the submaxillary, occipital, and cervical glands. This is usually absent in idiopathic roseola, although moderate general adenopathy may occur.

Treatment.—Macular erythema requires no external treatment beyond dusting powders and bland emollient applications. Its internal treatment is the treatment of the underlying conditions.

SCARLATINIFORM ERYTHEMA[1]

SYNONYMS: *Erythema scarlatinoides, Erythema scarlatinoides recidivans; Roseola scarlatiniforme* (Bazin), *Desquamative exfoliative erythema, Erythema scarlatinoides recidivans.*

The term scarlatiniform erythema is applied to forms of macular erythema which are of intense type resembling scarlet fever, and are followed by abundant or complete desquamation. It is rare.

It is an intense macular eruption of scarlatiniform type accompanied by somewhat more constitutional reaction than is usual in idiopathic erythemas and with more abundant exfoliation of the epidermis. In the very severest forms of this condition there is really a universal superficial dermatitis like confluent scarlatina. The inflammatory induration, while slight, is still apparent, and in the creases of the body, like the flexures of the joints and the folds of the neck, the epidermis may be thrown off and weeping fissures be formed.

Scarlatiniform erythema is always an abundant generalized eruption, and is usually universal, leaving no unaffected skin. It appears suddenly, reaches its full development in a few hours to a day, and begins quickly to subside. Exfoliation of the epidermis begins while the process is acute, and continues for a week or ten days, and is always abundant. Intense forms of the eruption are accompanied frequently by exfoliation of large sheets of epidermis. Occasionally a mask of an entire part, like a hand, will come away intact. After the first peeling in sheets, there is a branny exfoliation which continues for several days. Rarely the nails may exfoliate and the hair be shed in part. As in scarlatina transverse ridges in the nails are frequently left as a trace of the disturbance. An attack may run its course in a week or ten days. The disease, however, is apt to relapse, and repeated outbreaks may lengthen its course to six or eight weeks.

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[1] Brocq, *Jour. Cutan. Dis.*, 1885, p. 225 (Bibliography).—Ohmann-Dumesnil, *ibid.*, 1890, p. 293 (Bibliography).—Elliot, *New York Med. Jour.*, January 11, 1890.—Blanc, *Jour. Cutan. Dis.*, 1893, p. 11.—Hartzell, *University Med. Mag.*, August, 1895.

The subjective symptoms are trivial. There is a feeling of slight tension or heat in the skin and a little itching. In the extreme type of the condition there is, as in nearly all conditions of universal dermatitis, treme susceptibility to cold, and the greatest difficulty the patients have in keeping warm. The constitutional symptoms are likewise moderate. ey may be absent, but there is usually a febrile disturbance of one or o degrees, occasionally a rise to 103° F. with correspondingly exag-rated constitutional symptoms.

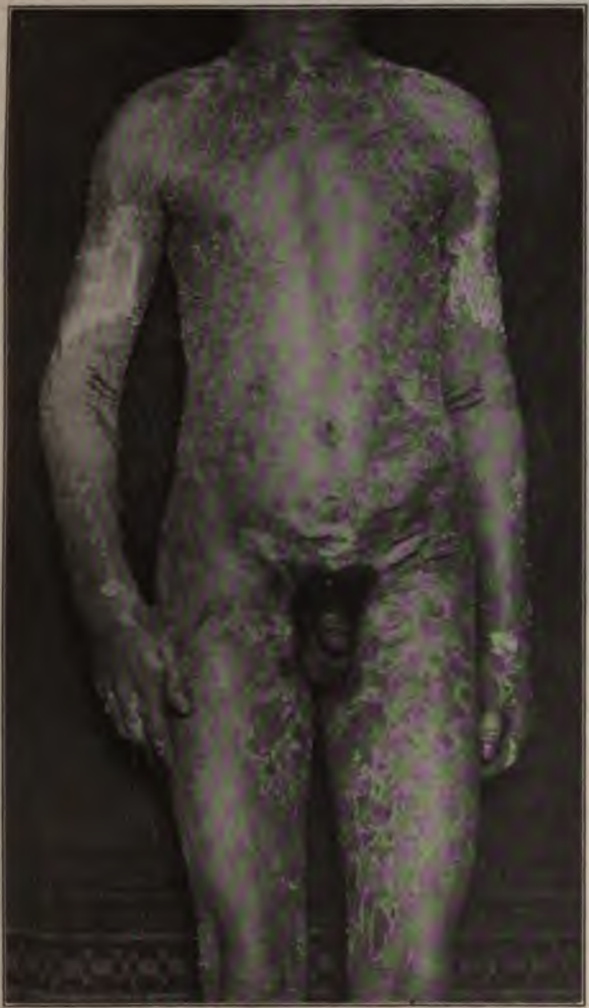

Fig. 30.—Erythema Scarlatiniforme. (Author's collection.)

Etiology.—The disease is in all probability due to the same sort of causes that produce the other forms of idiopathic roseola. It is a toxic disturbance. Personal idiosyncrasy probably plays a rôle in its production, for repeated attacks are apt to occur in an individual. It is most frequent in adults and in men. It is rare.

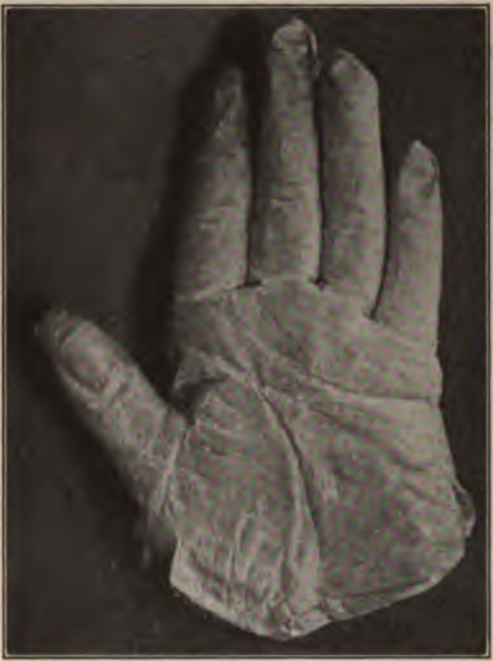

FIG. 31.—EPIDERMIC GLOVE, FROM SCARLATINIFORM ERYTHEMA. (Whitehouse.)

Diagnosis.—The disease can only be confused with scarlatina, from which it is distinguished by the absence of the characteristic severe constitutional and throat symptoms, and its shorter and less regular course.

Treatment.—The internal treatment consists of measures to provide for free elimination and to overcome any disturbances of the general health which may be found.

All that is necessary in the local treatment of scarlatiniform erythema is the application of bland dusting powders or the inunction of the skin with bland oils. In the extreme type of scarlatiniform erythema, where there is abundant exfoliation, the feeling of tension and stiffness is relieved and exfoliation is expedited by anointing the skin freely with a bland soft ointment, like boric acid (six per cent) in vaseline or with an oil like olive oil with boric acid (three per cent); instead of the boric acid, one half to one per cent carbolic acid may be used. The most important point in the care of these cases is rest and protection from cold, only obtainable by confinement to bed in a warm room. The patient suffers extremely if not protected against cold.

KERATOLYSIS

This disease, somewhat analogous to the cases of relapsing scarlatiniform erythema, is the rare condition of *deciduous skin* or *keratolysis*, in which the individual sheds his skin, like a snake, at more or less definite intervals. Various cases of this sort have been recorded.

In some the shedding of the skin occurs every month or six weeks, in others at longer intervals, and in some cases at a fixed time annually, as in the case of Frank and Sanford of Chicago. This phenomenon is usually observed first in early childhood, and occurs at intervals during the rest of life. The shedding of the skin occurs in large sheets, coming off at times like a glove or stocking. The nails are usually more or less affected in the attacks, and may be shed. The hair is less frequently involved. The onset of the condition is marked by more or less febrile and constitutional disturbances, followed by hyperæmia of the skin, which continues for twenty-four or thirty-six hours or longer. Exfoliation begins on the second or third day, and is complete in from three to ten days.

This condition is apparently a congenital anomaly, so that the resemblance to recurrent scarlatiniform erythema is clinical rather than etiological.

THE EXANTHEMATA[1]

The eruptions of scarlet fever, measles, and German measles are symptomatic erythemas. Their consideration, accordingly, as far as they are dermatological affections, should logically be taken up here, although it would be absurd to lose sight of the fact that they are con- titutional diseases of which the cutaneous efflorescences are but single eatures. The same is true of the eruptions of typhus and typhoid fevers, ut in these the eruptions are details of such minor importance that ey do not warrant consideration in a work on diseases of the skin. mallpox and chicken pox are also diseases which for logical sequence ust be included in the group of the exanthems, although, excepting eir prodromal macular rashes, they cannot by any stretch of the mean- g of the term be called roseolas. Their eruptions are inflammatory ions, the mechanism of whose production is probably the same as that the exudative erythemas, which are to be considered next. In the nsideration of the exanthemata here we are concerned only with the ptions and such other features of the disease as have a bearing on gnosis.

The exanthemata, while differing among themselves, possess certain mon features. They are specific infectious diseases, each one with a racteristic systemic disturbance and eruption. Each disease runs a nite and fairly regular course, and is self-limiting. They are all tagious, occur in epidemics, and are most frequent in childhood, because, except in the case of smallpox since the era of vaccination, th are so common that it is exceptional for an individual to pass throu childhood without exposure to them. One attack as a rule gives imm nity for life, but very exceptionally second attacks of any of them m occur. Any two of them may occur together. There has been repor the simultaneous occurrence of measles and scarlatina, measles and va ola, scarlatina and varicella, and vaccinia and each of the others. Wh two of them occur together they do not produce a new clinical pictu but each disease exhibits its independent features.

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[1] Welch and Schamberg, "Acute Contagious Diseases," Lea Bros. & Co., 1905.— ett, "The Exanthemata," F. A. Davis Co., 1902.—J. E. Graham, Articles on nthemata, Morrow's "System."

Their eruptions are all of toxic origin. They are symmetrical, m or less general, and each shows a characteristic distribution. The ext of the eruption varies enormously. It may be practically or actu absent, in very rare instances, in any of them; or in measles, sca fever, and smallpox it may be universal. Hæmorrhagic and urtica lesions may occur with any of them, an indication of their angioneur character.

I.—SCARLET FEVER

SYNONYMS: *Scarlatina, Scarlet Rash.*

Incubation Period.—The incubation period of scarlet fever, that of German measles and chicken pox, is variable. It probably a ages five or six days, and three to twelve days may be regarded as ordinary limits. Osler states that in a case where he undoubtedly car the contagion the incubation period was twelve days. It is, as a rule, than that of measles and smallpox.

Invasion.—The onset of scarlet fever may be preceded by a sl malaise. Whether this occurs or not, the onset begins suddenly. U ally without, sometimes, however, after a chill, the temperature rap rises, reaching on the first day 103° to 105° F. The pulse is full rapid. There is headache, nausea, and perhaps vomiting, and int thirst. In children convulsions are common. The face is flushed, skin is very dry, and on account of the dryness feels unusually hot. throat is dry and soon becomes congested and tender. The tongu covered with a gray fur, through which the filiform papillæ, swollen red, project. The duration of the stage of invasion is from twelv thirty hours, occasionally three days. In the severest cases the evide of a violent intoxication are very marked; in addition to the heada and vomiting there may be delirium, convulsions, and at times co These symptoms may be so severe that the patient dies in the prodro stage, overwhelmed by the poison.

Eruption.—Usually on the second day of the disease the erupt appears, first on the sides of the neck and on the chest, and then over rest of the body. The face may escape. Ordinarily the lesions do not appear around the mouth and nostrils, but leave an area which, on account of the intense suffusion of the surrounding skin, seems abnormally pale. The spread of the eruption is rapid, and within a few hours it reaches its full extent. It increases in intensity for perhaps twenty-four hours and persists for two or three days, when it begins gradually to fade. Occasionally it lasts only a few hours. The eruption consists of pin-head or slightly larger macules of a bright to deep scarlet red, which are so closely aggregated that they give the skin a distinctive diffuse scarlet color, more intense than is seen in any other of the exanthemata. Usually the diffusion of the rash is general, but occasionally it occurs in patches separated by areas of healthy skin. The color disappears on pressure, and, on account of the intensity of the surrounding red, the white line produced by drawing the finger over the surface is striking. At the height of the eruption there is perceptible swelling of the skin. Occasional petechiæ are not uncommon, and a frequent

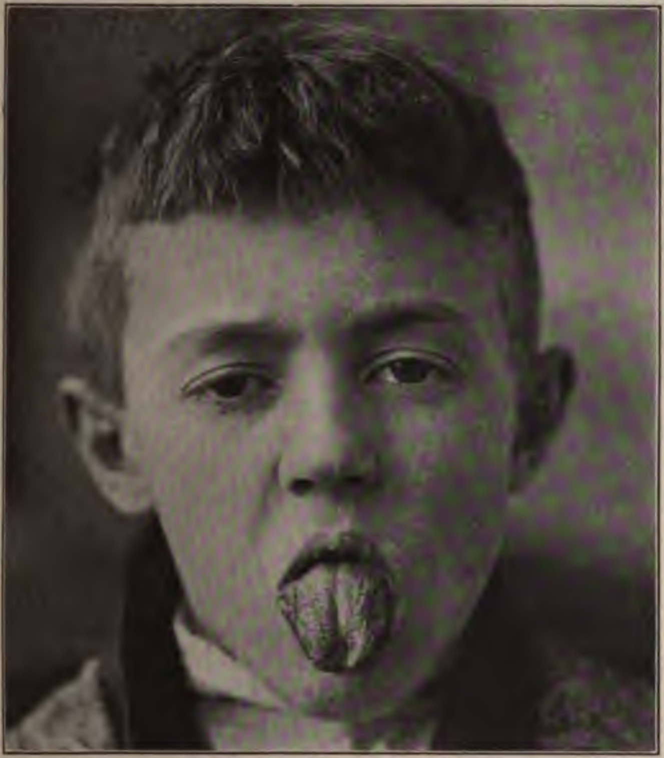

FIG. 32.—ENLARGED PAPILLÆ OF TONGUE IN SCARLET FEVER.
(Schamberg's collection.)

complication of the eruption is the appearance of sudaminal vesicles, especially about the flexures. They appear as minute translucent vesicles which soon become turbid. There may or may not be itching with the eruption, but there is ordinarily a feeling of tension and heat in the skin.

Variations in Eruptions in Scarlet Fever.—In exceptional cases the eruption of scarlatina may last only a few hours, in other cases it may last for nine or ten days. In still other cases the eruption becomes faint after the first day, and later again develops to the full intensity. As in measles, the eruption may in rare instances be abortive, being so faint that it is not observed, or not appearing at all, *scarlatina sine eruptione.* As, however, even in these cases desquamation is said to take place, it is probable that the eruption really occurs, but is so faint as to escape observation. In these cases the angina occurs and the ordinary complications arise. The diagnosis is only possible from a history of contagion or in epidemics.

Hæmorrhagic Scarlet Fever.—Hæmorrhage may occur in the lesions of scarlet fever, converting the picture into that of a dusky or livid hæmorrhagic exanthem. Extravasation of blood occurs in the mucous and serous membranes with discharge of blood from the orifices of the body. The hæmorrhagic forms of all the exanthemata are extremely grave, indicating the most profound intoxication; and in them the prognosis is very unfavorable.

The eruption may frequently be seen upon the mucous membrane of the mouth as closely set red points which cause a diffuse redness of the parts. The tip and borders of the tongue are red and its center covered with gray fur through which the swollen red papillæ protrude. Before the stage of eruption is passed the tongue begins to desquamate, becoming then of a deep red color with swollen papillæ. During the prodromal stage the pillars of the fauces and tonsils are engorged and reddened, with deep red points on the tonsils around the mouths of the follicles. As the disease becomes fully developed the throat symptoms become more marked. At its maximum the angina varies greatly. It may amount at the most only to swelling of the mucous membrane of the pharynx with more or less follicular tonsillitis, or there may be an intense inflammatory process involving to a greater extent the subjacent structures. Frequently the tonsillitis is severe; the follicles are distended with yellowish white exudate which forms a pseudo-membrane upon the surface. There may be ulceration, abscesses, and gangrene of the tonsils. In the severer types of angina the glands in the neck are engorged, and there is more or less inflammatory induration of the adjacent tissues.

The constitutional symptoms do not, as in smallpox, subside with the appearance of the eruption, but persist during the stage of eruption and adually disappear as the eruption fades. The temperature curve of rlet fever is an important diagnostic feature. There is a very rapid e on the first day. The fever continues high with slight daily remis- n during the period of eruption. If the angina is slight and there e no complications the temperature falls with the fading of the erup- n. It may reach normal on the fourth or fifth day. The duration of e fever, however, depends upon the extent of complications, gradually subsiding usually as the throat symptoms disappear. In severe cases it may continue to the ninth or twelfth day.

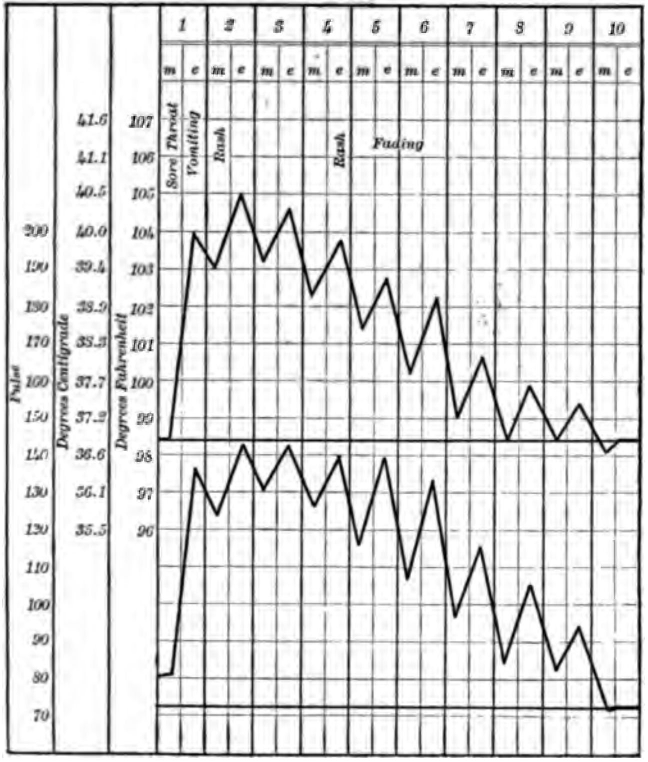

Fig. 33.—Temperature Chart of Scarlet Fever. (Butler.)

Desquamation.—Upon the disappearance of the eruption the skin is slightly roughened and desquamation soon begins. It is more abundant than in any of the other exanthemata, but its intensity varies in a general way with the intensity of the eruption. It is usually in coarse scales with exfoliation of masses of epidermis upon the extremities where the horny layer is thickest, but it may come away in large sheets after an intense eruption, and even as a mold of an entire hand or foot. It lasts longest upon the hands and feet. In extremely rare cases the nails are thrown off, and it is common for transverse ridges to persist in the nails for some time afterwards as an evidence of the interference with their nutrition. Desquamation continues ordinarily from ten days to three weeks. It may continue for six or seven weeks.

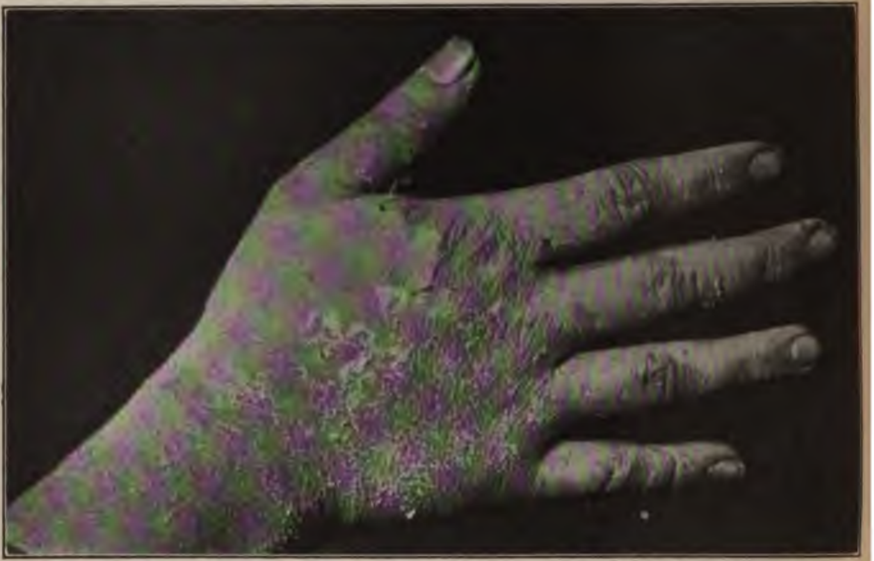

FIG. 34.—DESQUAMATION IN SCARLET FEVER. (Schamberg's collection.)

II.—MEASLES

SYNONYMS: *Morbilli, Rubeola.*

Incubation.—The incubation period of measles, like that of smallpox, is fairly uniform, and is usually from ten to twelve days. It may be somewhat shorter, and in extreme cases may be eighteen to twenty days.

Invasion.—The onset of measles is not so sudden as that of scarlet fever. The disease begins like a severe cold, and the symptoms increase through the second and third day until the appearance of the eruption. There are at first the usual symptoms of febrile disturbance with marked evidence of involvement of the mucous membranes. There are sneezing and coryza, injection of the conjunctivæ, lachrymation, and cough. The temperature may rise rapidly on the first day to 102° or 103° F., but usually the fever increases from day to day during the stage of invasion, reaching 103° to 104° or 105° F. by the time of the appearance of the eruption.

During the stage of invasion, ordinarily on the second day, there is evidence of the eruption of measles in a punctate rash upon the mucous membrane of the mouth. This eruption has been long recognized, but it has been left for Koplik in 1896 to describe a pathognomonic lesion, the so-called Koplik's spots, which may be seen upon the mucous membrane of the mouth as much as seventy-two hours before the appearance of the eruption upon the body. This eruption is, in his words, as follows: "If we look in the mouth at this period (invasion) . . . on the buccal mucous membrane and inside of the lips, we invariably see a distinct eruption which consists *of small irregular spots of a bright red color. In the center of each spot there is noted, in strong daylight, a minute, bluish-white speck.* These red spots with accompanying specks of a bluish-white color are absolutely pathognomonic of beginning measles, and when seen can be relied upon as a forerunner of the eruption. . . . Sometimes only a few red spots with this central bluish point may exist —six or more; in marked cases they may cover the whole inside of the buccal mucous membrane.

"The eruption just described is of greatest value at the very outset of the disease—the invasion. As the skin eruption begins to appear and spreads, the eruption on the mucous membrane becomes diffuse and the characters of a discrete eruption disappear and lose themselves in an intense general redness. When the skin eruption is at the efflorescence the eruption on the buccal mucous membrane has lost the character of a discrete spotting, and has become a diffuse red background with innumerable bluish-white specks scattered on its surface." The eruption in the mouth fades before that on the body. The distinctive character of this lesion has been confirmed by many observers. It is not invariably present, but is found in over ninety per cent of cases.

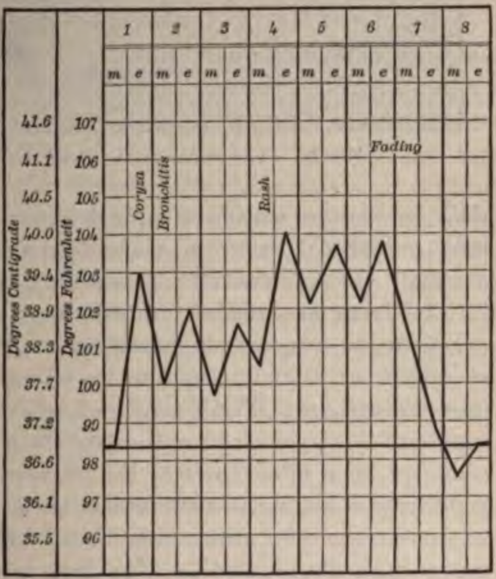

FIG. 35.—TEMPERATURE CHART OF MEASLES. (Butler.)

The stage of invasion ordinarily lasts about three days. In some cases it may be prolonged to five or even six or seven days.

Eruption.—As a rule, on the fourth day the eruption appears, first on the face, usually on the forehead or cheeks, thence it passes down over the body and becomes generalized in twenty-four to forty-eight hours. The eruption of measles is most abundant upon the face, and is least abundant upon the lower extremities. The eruption persists for two or perhaps three days, and then gradually fades. It consists of maculo-papules which are slightly elevated. The lesions are ordinarily larger than those of scarlet fever, but vary in size from a pin head to a finger nail. The size of the individual lesions increases considerably after their appearance. They are irregularly roundish in outline and their borders are usually sharply defined. They are not simple macules, but are distinctly elevated maculo-papules, and where the eruption is abundant there is considerable swelling of the skin. The lesions vary in color from deep reddish to dusky red; they are of a good deal darker red than the eruption of scarlet fever. The rash usually shows as an abundant but discrete eruption, the lesions frequently being arranged in crescentic groups. The lesions may be more or less confluent about the face, and in rare instances the eruption is so abundant as to be almost confluent upon other parts of the body. As in scarlet fever, occasional petechiæ are not uncommon and miliaria is frequent.

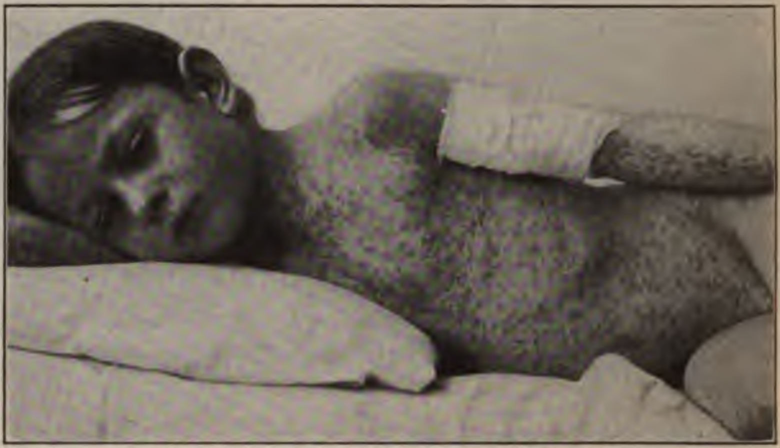

FIG. 36.—ERUPTION OF MEASLES. (Schamberg's collection.)

In certain very grave cases extravasation of blood into the lesions occurs with the production of a dark mahogany red or black hæmorrhagic eruption, *hæmorrhagic measles.* In these cases extravasation of blood is likely to occur into the mucous and serous membranes with discharge of blood from the orifices of the body, and, as in all of the exanthemata, when this condition occurs the prognosis is very grave. In hæmorrhagic measles the hæmorrhage usually occurs before the eruption is fully developed.

It is well established that during epidemics of measles patients in rare instances show the prodromal symptoms of the disease, but convalesce without the appearance of the eruption—*morbilli sine eruptione.* Occasionally the eruption of measles fades before it is fully developed, and later regains its intensity. This is usually due to cardiac weakness from severe pulmonary involvement, and the phenomenon therefore is largely a mechanical one, from failure of the circulation in the skin, and is of unfavorable import.

Desquamation.—After the eruption has persisted for two to three days it gradually fades, the constitutional symptoms subside, and by the eighth or ninth day of the disease the eruption has disappeared and desquamation begins. The amount of this varies with the intensity of the eruption. In all cases it is much less than in scarlet fever. It occurs in fine branny scales which may not be observed at all. It is usually most noticeable about the face. It ordinarily continues only for a few days, but may persist for ten days or more.

III.—GERMAN MEASLES

SYNONYMS: *Rubella, Rötheln, Epidemic Roseola.*

German measles is an acute contagious exanthem which resembles in some of its features both measles and scarlatina. It manifests to a slight degree the involvement of the mucous membranes of measles and the angina of scarlet fever. Both of these features, however, are very much less intense than in the other diseases, and either may occur without the other. The eruption ordinarily presents similarity to that of measles; in rare cases it imitates closely that of scarlet fever. German measles, however, is an independent specific disease: its occurrence protects the individual from subsequent attacks, while it affects and runs a normal course in individuals who have previously had measles and scarlet fever.

Incubation.—The incubation period is variable, from five to twenty days. This variability contrasts with the fairly definite incubation period of scarlet fever and measles.

Invasion.—The symptoms of invasion are ordinarily very mild, so mild that they often escape notice. There is, as a rule, slight feverishness, a temperature from 99° to 100° F., with the usual symptoms accompanying a slight febrile disturbance. In rare instances the prodromal symptoms are severe with temperature of 101° to 103° F., coryza, hoarseness, sore throat, cough, suffusion of the eyes, nausea and vomiting, headache and backache, and even delirium, but such symptoms are so exceptional that they would entirely mask the disease. The stage of invasion is very short, usually not more than half a day. It may, however, be prolonged to three to five days in rare instances, the longer stage of invasion presaging a severe manifestation of the disease.

Eruption.—The eruption usually occurs first on the face, but almost at the same time or very soon afterwards it develops on the upper part of the trunk and arms. It spreads rapidly, becoming generalized in twenty-four to forty-eight hours. Individual lesions begin to fade within a few hours, and in its development the eruption comes out in crops and not continuously. As a consequence of this, while the later lesions are appearing the earlier lesions are fading, and so lesions in all stages of their course may be seen. The duration of the eruption is usually short. It may have faded almost within twenty-four hours of its appearance. Occasionally it persists as long as three days, and in very rare cases four to six days. The duration of the individual lesions and of the entire eruption is in proportion to the severity of the attack.

The eruption consists of pin-head- to finger-nail-sized macules which are very slightly if at all elevated, not so much so as the lesions of measles. They are roundish or irregular, of fairly well-defined outline, and of a bright pink color. They have not the vivid color of scarlet fever nor the dark red tinge of measles. The lesions are commonly discrete and not so abundant as those of measles. Upon the face and other parts where the lesions are most abundant they may coalesce into red patches. As a rule, the grouping in crescentic figures seen in measles does not occur. The eruption, like that of measles, is most abundant upon the face, where it may produce slight swelling. There is not the pale area around the mouth and nostrils seen in scarlet fever. Over the rest of the body it is generalized, being most sparse, as a rule, below the knees. The palms and soles may be involved. In rare instances miliaria complicates the eruption, and petechiæ have been observed.

As is evident from the foregoing, the eruption usually resembles that of measles, and in severe cases may simulate measles so closely that the diagnosis from the eruption alone is impossible. In rare cases rubella occurs as an abundant erythematous rash of pin-head-sized lesions which closely simulates that of scarlet fever. It may be so abundant as to produce a uniform redness, as in scarlatina.

At the time of appearance of the eruption there may be considerable itching; ordinarily this is evanescent, and is the only subjective symptom associated with the skin.

The constitutional symptoms are, as a rule, trivial. The temperature does not rise above 100° F.; there is some lachrymation, coryza, sneezing and cough, and congestion and tenderness of the throat. These are all much less marked than in measles and scarlet fever. In the very severe cases the temperature may reach 103° or 104° F. with corresponding intensity of other symptoms, but this is excessively uncommon.

esquamation.—Desquamation is very slight, and in mild cases ually not seen; in severer cases it occurs as a slight furfuraceous ng. It is most likely to be noticeable on the nose.

iagnosis.—The most striking contrast from measles and scarlet is in the mildness of the constitutional symptoms. The absence of

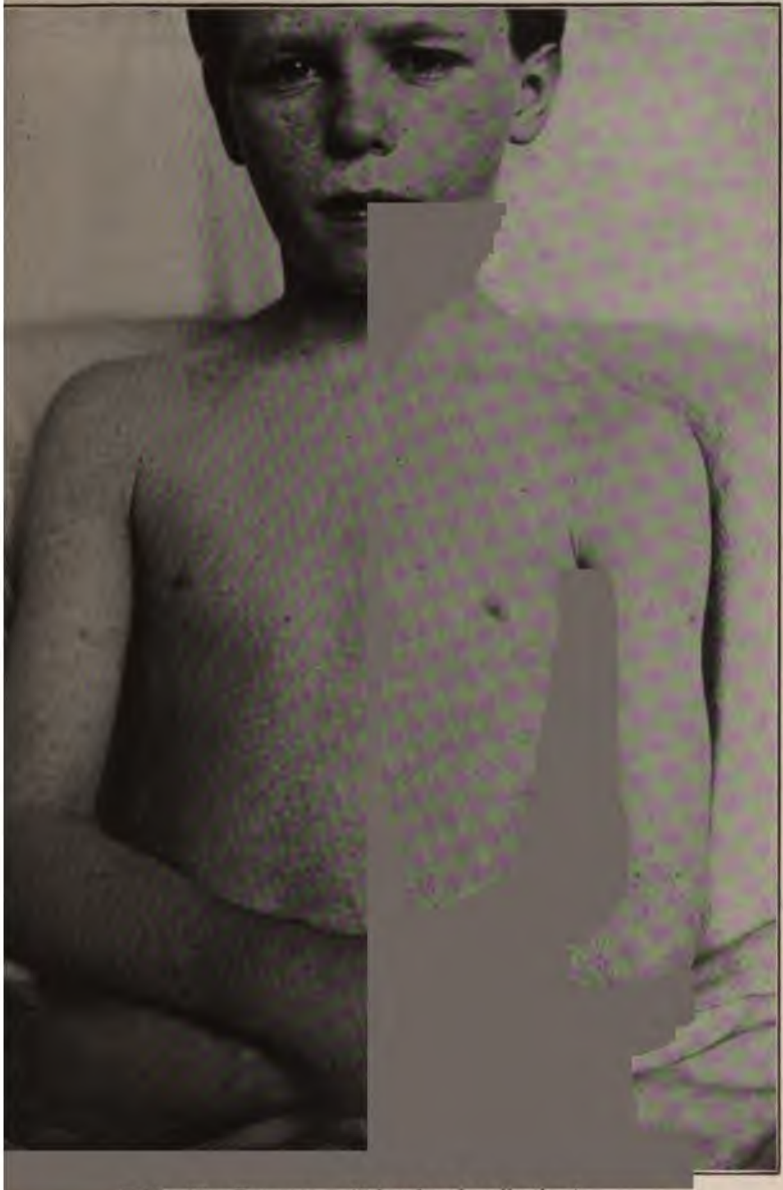

FIG. 37.—RUBELLA. (Schamberg's collection.)

severe catarrh or angina, the enlargement of the postauricular and post-cervical glands, the faintness of the eruption and its polymorphous character—varying in age at different points—and the clinical history, all serve to differentiate it readily from measles and scarlet fever. It is most likely to be confused with idiopathic roseola. In rubella the peculiar glandular involvement, the presence of some angina and catarrhal symptoms, and especially the history of contagion are the chief points in diagnosis from idiopathic roseola.

DIFFERENTIAL DIAGNOSIS OF THE MACULAR EXANTHEMATA

	SCARLET FEVER.	MEASLES.	RUBELLA.
Incubation.....	3–7 days.	10–12 days.	5–22 days.
Invasion.......	1–2 days.	3– 4 days.	½– 1 day.
Eruption.......	3–4 days.	3– 4 days.	1– 3 days.
Desquamation..	2–4 weeks.	5–10 days.	2– 3 days.
Eruption.......	1st on side of neck and front of chest; spares nose and mouth.	1st on forehead and face.	1st on face or upper half of body.
	Pin-head macules; bright scarlet; closely aggregated causing diffuse erythema.	Macules or maculo-papules much larger than in scarlet; dark reddish color; usually discrete and less abundant than in scarlet; crescentic arrangement.	Pinkish red macules of the size of those of measles.
Desquamation..	Large scales and sheets; abundant.	Fine branny scales; slight.	Fine branny scales; very slight.
Temperature ...	Rises suddenly; remains high; 102°–105° F.	Gradual, reaching with appearance of eruption 103°–106° F.	99°–100° F.
Symptoms......	Angina without catarrh.	Catarrh of the respiratory and ocular mucous membranes without angina.	Slight catarrh and angina, or either.
	Tongue thickly coated; with swollen red papillæ.	Thickly coated tongue.	Slightly coated tongue.
	Severe constitutional disturbances.	Constitutional disturbances less severe, chiefly of the respiratory system.	Slight constitutional symptoms.
Koplik's spots...	Absent.	Present.	Absent.
Complications...	Tonsillar abscesses, adenitis, otitis media, nephritis, arthritis, sepsis.	Bronchitis, pneumonia, subsequently tuberculosis.	No characteristic complications.

In scarlet fever, and less frequently in measles, there is some general adenopathy. This is more common in rubella, and a fairly constant symptom in rubella is enlargement of the postauricular, suboccipital, and postcervical glands, which increase during the eruptive stage. The enlargement of these glands is a fairly characteristic diagnostic feature of rubella.

IV.—SMALLPOX

SYNONYM: *Variola.*

Incubation.—Variola, when communicated by contagion, has an incubation period of from ten to fourteen days; if inoculated, the period is somewhat shorter, eight to nine days. After this period of incubation there occurs occasionally a moderate rise of temperature, 100° or 101° F., for one or two days before the acute onset of the disease, but usually there are no symptoms during the incubation period.

Invasion.—As a rule the onset of the symptoms of smallpox is sudden and intense. There is a severe chill or series of chills, with rapid rise of temperature to 104° or 105° F., headache of the most intense character, severe backache most marked in the lumbar region, pain in the legs, and vomiting. This combination of symptoms—sudden severe chill, followed by high temperature and intense headache and backache—should always put one on his guard when smallpox exists, and especially in patients who have not been vaccinated. With these classical symptoms the patient is exceedingly nervous, and delirium or convulsions may occur. The pulse is full and rapid, 110 to 130, 140 to 160 in infants. There is a dry, furred tongue with some angina, catarrh of the respiratory mucous membranes, and suffusion of the face and eyes. The constitutional symptoms increase in intensity until the eruption appears. The vomiting may cease, but the headache and backache persist.

Prodromal Eruptions.—During the stage of invasion there occurs in some of the cases an erythematous eruption which is quite independent of the typical eruption of variola. This prodromal eruption occurs in two forms: as a scarlatiniform or as a macular morbilliform eruption. With either, hæmorrhagic macules occur, and in very rare instances urticarial wheals. This eruption is usually situated over the lower part of the abdomen, the lateral thoracic, and the axillary regions; it also occurs occasionally on the flexor surfaces of the larger joints and less frequently on the extensor surfaces of the knees and elbows. In some cases it is as generalized as the eruptions of scarlet fever and measles, and may be almost indistinguishable in appearance from them. It appears upon the second or third day of the stage of invasion, and either fades out or is lost on the appearance of the true eruption. This eruption most frequently resembles that of measles, but it lacks the characteristic distribution of the eruption of measles, is of short duration, disappearing in the course of twenty-four to forty-eight hours, and the lesions are without the elevations of those of measles. Sometimes the prodromal eruption is distinctly hæmorrhagic rather than scarlatiniform or morbilliform. It then occurs in the form of grouped, pinpoint- to pinhead-sized, dark red to purplish hæmorrhagic macules, which may be so abundant as to produce a diffuse redness of the skin. The prodromal eruption is said to be, in rare instances, the only cutaneous manifestation of the disease.

The frequency of the prodromal eruptions varies in different epidemics. They occur in from ten to sixteen per cent of cases, according to Osler. Graham observed them in two of seven cases. According to Hyde they are most frequent in children and menstruating women. The recognition of this prodromal eruption is of great importance. Its occurrence, together with the classical constitutional symptoms, may be a valuable fact in confirmation of one's suspicions in the early period of a doubtful case, and its recognition is equally important to avoid confusion with the other exanthemata.

Eruption.—After the constitutional symptoms have continued from two to four days in severe cases, and from four to five days in mild cases, the typical eruption appears; and with its appearance there is rapid improvement in the constitutional symptoms. The temperature falls, the headache and backache cease, and in mild cases the patient may no longer feel that he is ill. The typical eruption of smallpox usually occurs about the fourth day, and appears first upon the head near the hairy border of the forehead and upon the wrists. Within twenty-four hours lesions develop upon other parts of the face, especially around the nose and mouth and eyes, on the scalp, and on the extremities, and a few appear on the trunk.

The eruption of smallpox goes through a definite evolution. At first it is papular; after one or two days each papule is converted into a vesicle; after two to three days more the vesicle is transformed into a pustule. The pustule exists as such for from about two to six days, gradually drying up. The time from the appearance of the papule to the shedding of the pus crusts is ordinarily about eighteen to twenty-four days. The eruption of smallpox does not appear all at once; an interval of thirty-six to forty-eight hours occurs between the appearance of the first and of the latest lesions, and their further evolution corresponds in time with the order of their development.

The rash appears at first as red papules of the size of a pinhead or larger, situated deep in the skin and peculiarly firm to the touch—feeling like shot imbedded in the skin. During the first two days of the eruption the papules increase in size and number, and about the second day they begin to be transformed into vesicles, a translucent vesicle form-g upon the summit of the papule. These vesicles are sharply elevated d umbilicated. The umbilication is marked, producing in many le-ons a peculiar puckered appearance. This umbilication, which is char-cteristic, occurs, it is to be remembered, in the vesicular stage, and is ot to be confused with the pseudo-umbilication which comes from the formation of a pus crust in the center of a lesion at a later stage of the

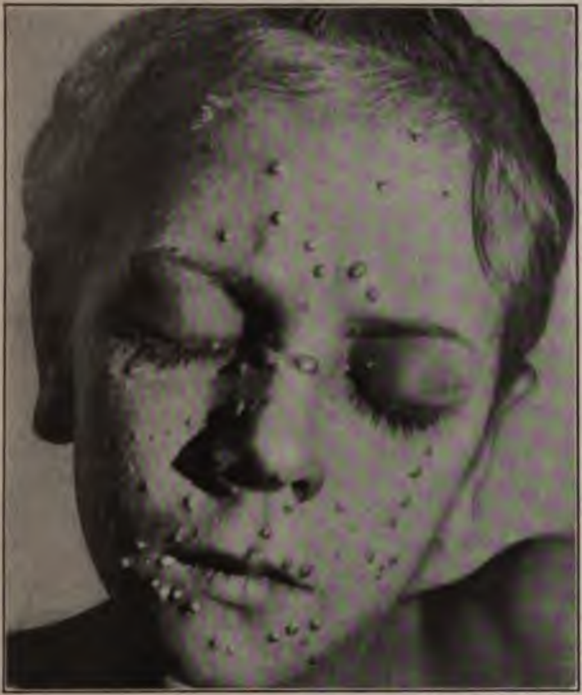

FIG. 38.—DISCRETE VARIOLA. (Schamberg's collection.)

eruption. The vesicles are at first clear with a characteristic hyaline appearance due to the thickness of their covering; the contents rapidly ecome milky, and within two days, by the sixth or eighth day of the isease, they become purulent. At this stage the lesions are the size of a ea or larger. They are surrounded by an angry red areola, and if osely aggregated the skin is much swollen and of a livid red color.

The fully developed pustule of smallpox is a characteristic lesion. It sharply elevated, pointed or globular—for the umbilication has disap-ared—is of a yellow color on account of its purulent contents, and situated on a firm, angry, red base. It is larger than most other pus-es and stands out sharp and aggressive from the skin. The pustules, er an existence of two or three days, begin to rupture, and by the tenth or eleventh day they begin to dry up, desiccation being well advanced in the earlier lesions by the fourteenth or fifteenth day. The pus first dries into yellowish, brownish, or blackish crusts which adhere closely and are not thrown off for several days. With the desiccation of the pus there is also desquamation of the horny epidermis, and entire molds of the hands or feet may come off intact. The stage of desiccation begins with the third week, and is usually completed by the end of the fourth week, but may continue for six weeks from the beginning of the disease.

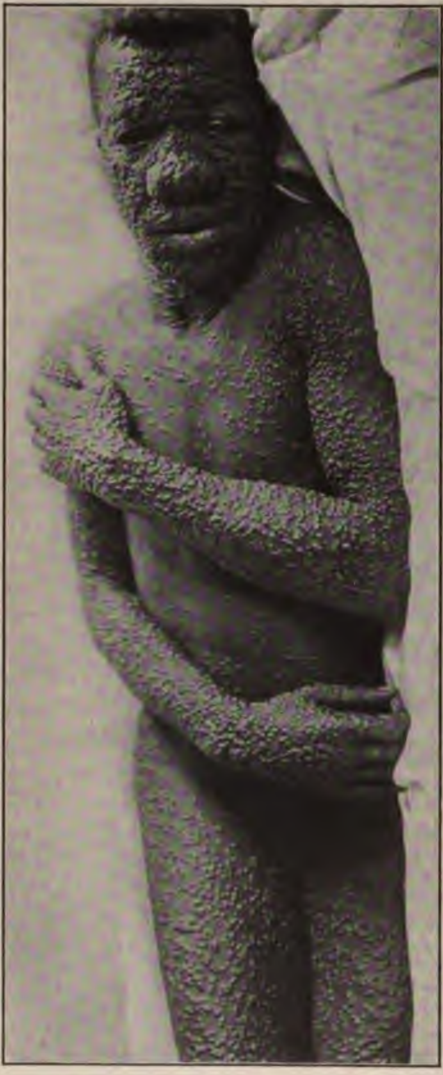

FIG. 39.—CONFLUENT VARIOLA, SEVENTH DAY OF ERUPTION. (Schamberg's collection.)

After desiccation and desquamation are complete there are left at the sites of the lesions purplish-brown pigmented spots which disappear slowly. As is well known, in severe smallpox there is much scarring. This is due to the fact that the pustules of smallpox are true abscesses. They involve the corium and cause complete destruction of connective tissue which can be replaced only by scars. The amount of pitting depends largely upon the severity of the disease. In discrete smallpox the pitting is very slight and may not be visible.

The eruption of smallpox occurs characteristically upon the face and extremities, including the palms and soles, and it is always most abundant in these localities. The extent of the eruption varies between the widest extremes. In the mildest cases the entire eruption may amount only to a few discrete lesions upon the forehead, around the nose, and upon the hands and feet. In the severe confluent cases the eruption is so abundant upon the head and extremities that the skin is honeycombed with abscesses. Upon the trunk, even in these most severe cases, the eruption is somewhat less abundant and is not quite confluent. In such cases at the height of the pustular stage, about the eighth to tenth day, there is enormous swelling of the skin. This is well marked over the entire surface, but is particularly striking in the distortion which it causes in the features. Along with this the pus, as it is freed from the ruptured pustules, dries on the surface into thick yellowish and, later, brownish or blackish crusts which cover the surface with a dirty suppurating mask. At the same time there is exhaled a characteristic and peculiarly offensive odor of suppuration. "At this stage the patient presents a terrible picture unequaled in any other disease; one which fully justifies the horror and fright with which smallpox is associated in the public mind" (Osler). Hardly less striking than this extreme picture of smallpox is the apparently benign picture which the disease presents in the mildest cases. And between these two extremes we see all degrees of intensity of the eruption.

Variations in Eruption. VARIOLA SINE ERUPTIONE.—Occasionally during epidemics patients present the prodromal symptoms of variola, perhaps have a prodromal rash, but the disease subsides at the time for eruption, and there appears no typical eruption, or at the most a few variolous papules which abort without even becoming vesicular. It is the mildest form of smallpox and is rare.

VARIOLOID.—Varioloid is mild (modified) smallpox, which occurs in persons who are relatively immune. This immunity may be either natural or the result of previous vaccination. When varioloid follows vaccination it is in cases in which the vaccination was not perfect or the immunity which it confers has partially failed. This same failure of immunity may occur even long after a previous attack of smallpox. The identity of varioloid with smallpox is shown by the fact that typical smallpox may be contracted from it.

In varioloid the prodromal symptoms are those of smallpox, and they may be severe; ordinarily they are mild. The prodromal rashes are common. The eruption occurs at about the same time as in variola, and with its appearance the constitutional symptoms subside and later constitutional symptoms do not usually occur. The papules which develop are few in number and are situated on the face and hands. The lesions pass through their evolution rapidly and may abort without undergoing the typical changes. The pustules dry up, the crusts are thrown off, and rarely does any scarring occur; it is even less than in varicella. *Variola sine eruptione* is the mildest form of varioloid.

Variola Hæmorrhagica.—Hæmorrhagic smallpox occurs in two forms. In the first, extravasation of blood into the skin occurs on the second or third day of the disease, similar extravasations occurring into the mucous membranes, accompanied by hæmorrhage from the orifices of the body. The lesions are of mahogany or purplish color, do not disappear on pressure, and may be so extensive as to be almost confluent. In these cases the patients are overwhelmed by the poison, and death usually occurs before the sixth day of the disease. In the second form of hæmorrhagic smallpox the hæmorrhage occurs into the vesicles or pustules with more or less extravasation into the substance of the skin, and the lesions are changed from their typical color to a dark brown or black. In both types of hæmorrhagic smallpox extravasation takes place upon the mucous membranes and hæmorrhages from the orifices of the body are common. The prognosis in both these forms of hæmorrhagic smallpox is extremely grave; the earlier the hæmorrhage occurs the graver the prognosis. In the first form death usually occurs by the sixth day; in the second form recovery in rare instances takes place.

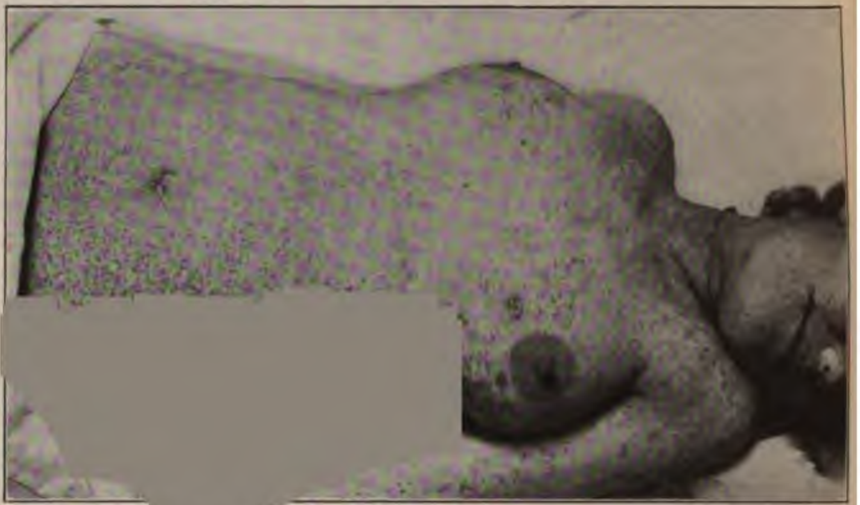

Fig. 40.—Hæmorrhagic Variola. (Schamberg's collection.)

Occasionally petechiæ occur with the eruption in cases which are not severe.

With the appearance of the eruption upon the skin it also appears to greater or less extent upon the mucous membranes. During the stage of invasion the pharynx is dry and congested and the conjunctivæ suffused. As the eruption develops upon the skin, lesions appear upon the mucous membranes. The tongue, the pharynx, and the larynx become reddened and swollen, and as the disease progresses more or less ulceration occurs, the extent of these involvements varying with the intensity of the eruption. There is more or less salivation; in severe cases the saliva becomes thick and tenacious and drools from the mouth. Suppurative glossitis, tonsillitis, and ulceration and gangrene of the fauces are frequent complications in severe cases, as are œdema and destructive ulceration of the larynx. The eyes early show conjunctivitis; lesions develop upon the conjunctivæ, and may appear also upon the corneæ and cause their destruction through ulceration. Before the days of vaccination smallpox was the greatest cause of blindness.

The intensity of the constitutional symptoms of the stage of invasion does not always bear a direct relation to the intensity of the disease in its eruptive stage. A very acute prodromal stage may be followed by a very mild eruptive stage, although this is not the rule. The constitutional symptoms, however, which accompany the eruptive stage correspond in intensity with the extent of the eruption. With the appearance of the eruption and during the papular and vesicular stages the constitutional symptoms may disappear and the temperature drop to normal, or there may be a defervescence of the symptoms without their complete disappearance. And this may be the end of the constitutional symptoms, so that it is not uncommon to find patients in the eruptive stage of smallpox going about and insisting that they are not ill. If the eruption is at all abundant, however, there develops with the pustular stage a suppurative fever. It varies in intensity with the extent of the suppurative process, and disappears with the disappearance of the eruption.

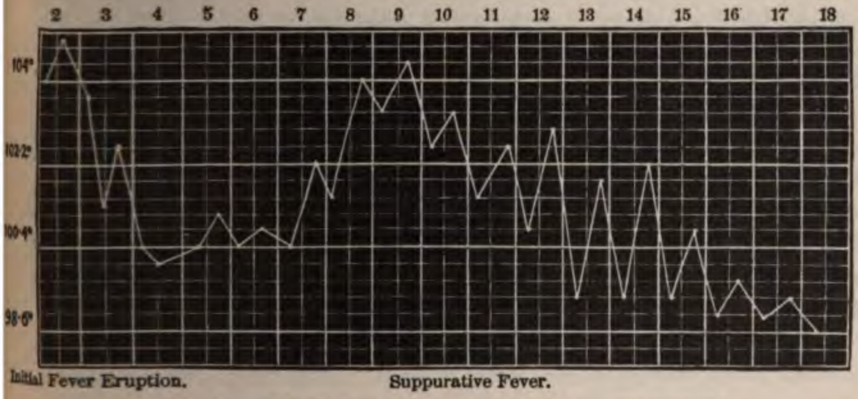

FIG. 41.—TEMPERATURE CHART IN VARIOLA. (Strümpell.)

Diagnosis.—The most important fact in the diagnosis of smallpox is the characteristic sequence of the disease: the history of sudden onset, with chills, high fever, severe backache and headache, and vomiting; th continuance of these symptoms for from three to five days, and thei subsidence upon the appearance of a shotty eruption, first upon th forehead. When the prodromal symptoms or the eruption are well marked there should be little trouble in diagnosis. Unfortunately it is not always easy to get a satisfactory history of the prodromal symptoms, and further it is not uncommon for the eruption and the later constitutional symptoms to be so mild as to be deceptive. Even in the mildest cases, however, the few lesions which do exist about the face and forehead and the hands and wrists present the peculiar characteristics of smallpox. They are very hard, shotty papules, they are umbilicated vesicles, or they are unusually large, prominent, peculiar-looking pustules with violaceous angry bases and projecting globular yellow apices.

The prodromal eruption of variola may be confused with measles o scarlatina. It may ordinarily be differentiated by the fact that it i not nearly so abundant. In some cases, however, the appearance of th eruption may be almost indistinguishable from that of scarlet fever o measles. In such cases diagnosis must depend upon the presence of th clinical history and the constitutional symptoms of smallpox, and upo the absence of these features of scarlatina and measles.

In its papular stage the eruption of smallpox, with the firm, re deep-seated, shotty papules, is unlike any other cutaneous eruption.

In the vesicular stage smallpox is most likely to be confused wi chicken pox. In chicken pox the eruption is more abundant upon th trunk than upon the face and extremities. The lesions are smaller an without the firm base of smallpox. The vesicles are superficial, co ered by a thin epidermal wall, and have the yellowish color of serum an not the hyaline appearance of the variola vesicle. On the palms wh the walls are thick the vesicles may appear like those of variola. Um bilication of the *vesicles* does not occur. The prodromal stage is short and lacks the characteristic features of that of smallpox, and constit tional symptoms are usually altogether slight.

In the pustular stage of smallpox a well-marked abundant eruptio is hard to confuse with any other disease. If, however, the lesions a everywhere discrete and are not abundant, confusion with a pustul syphilide, or even an indurated acne, is quite possible. In such cases th marked difference in the clinical history is especially important. Th lesions themselves also usually present sufficiently characteristic diffe ences. The eruption of smallpox is a uniform pustular eruption, th lesions are large, sharply elevated and conical or globular. In a pustul syphilide the eruption is not uniform; there may be a preponderance pustules, but other lesions will also be found—scaling papules, fre papules, fresh pustules, and old pustules which have dried up and which desiccation is already far advanced. In other words, the eruption has not come, like that of smallpox, as one crop, but has developed or is appearing from time to time irregularly, and lesions can be found in all stages of development. Along with the pustular syphilodermata also there are found mucous patches in the mouth or about the genitals, and quite probably also flat ulcerating papules around the anus. In almost all of these cases traces of a recent initial sclerosis can be found upon careful search. The lesions of indurated acne, with their centers occupied by comedones, and with independent comedones in the skin, present only a superficial resemblance.

V.—VACCINIA

Synonyms: *Vaccination, Cowpox.*

Vaccinia is the term applied to the eruption which is produced by the artificial inoculation of cowpox in the human subject. Vaccinia is probably a manifestation of smallpox whose virus has been altered by development in an animal which, while susceptible to smallpox, is much less susceptible than man and in which the disease is reproduced in a greatly ameliorated form.

After a primary vaccination there occurs on the third or fourth day a redness at the point of inoculation, upon which a papule quickly develops. The papule increases in size, and on the fourth to seventh day, usually the fifth, becomes converted into a small, round, slightly umbilicated vesicle the size of half a wheat grain, situated upon an inflamed base. The vesicle increases in size until the eighth day, and the umbilication becomes more marked. It then persists for about two days as a round distended vesicle with clear or milky contents on a hard inflamed base. By the eleventh or twelfth day the inflammatory reaction begins to subside, the contents become turbid or purulent, the vesicle becomes flaccid and begins to desiccate. Within fourteen days a brown crust forms which at the end of twenty-one to twenty-five days separates, leaving a small pitted scar.

Usually the inoculation is made at several adjacent points, so that instead of one vesicle there develop several vesicles whose inflammatory bases coalesce, forming a finger-nail-sized or larger lesion with an inflammatory base upon which are several discrete vesicles that ultimately become purulent and dry into a crust.

In rare instances extravasation of blood occurs in the areola of vaccination, *vaccinia hæmorrhagica.* This may be associated with other purpuric symptoms.

Occasionally spontaneous vaccine vesicles develop around the point of inoculation.

When the vaccination lesion becomes well developed, swelling of the neighboring lymphatic glands is likely; of the axillary glands when the vaccination is done upon the arm; of the inguinal glands when upon the leg.

With the development of the first papules there is often considerable itching, which persists until the lesion shows well-marked vesicles. When the vesicles are fully developed and the inflammation at the base greatest the lesion is throbbing and painful. On the third or fourth day of the eruption there is likely to be a rise of temperature to 100° or 101° F., with more or less malaise. This may disappear quickly or persist until the eighth or ninth day. It is usually a trivial reaction.

The scar which follows a successful vaccination is characteristic. It is a roundish, sharply defined, white scar, somewhat depressed and frequently more or less atrophic, showing over the surface conical pittings.

Generalized Vaccinia.—Generalized vaccinia may be local or constitutional. Local generalized vaccinia is due to repeated inoculations, the vaccinia reproducing itself at each point of inoculation.

Generalized vaccinia from systemic infection is exceedingly rare, but does occur. In this the vaccinal eruption is reproduced in different parts of the skin from the distribution of the virus through the body. It has been caused by the ingestion of vaccine material and the production of general vaccine infection in that way. The explanation of its occurrence probably lies in unusual susceptibility of the individual; the inoculation of cowpox in such individuals producing not simply a local but a generalized cowpox infection. The condition may be compared to varioloid: generalized vaccinia being the product of an attenuated virus in an individual of low resistance, varioloid the result of a normal virus in an individual of extraordinary resistance.

In generalized vaccinia of systemic origin the lesions appear in crops and pass through the evolution of vaccinal lesions. The number of lesions may be few or abundant. The eruption appears from the sixth to the ninth day after vaccination, and disappears about the twentieth day. There is usually some systemic reaction, and it may be severe.

It is to be noted that vaccination passes through its active stage by the end of about fourteen days; in other words, it produces its constitutional effects within a period which corresponds approximately to the period of incubation of smallpox. It is this short period of incubation and the comparatively rapid course which it pursues which renders vaccination so valuable as a preventive measure after exposure to smallpox.

Variations.—Primary vaccination usually follows the typical course. Occasionally, however, primary vaccination and more frequently revaccination pursue an atypical course. Vesiculation on the one hand may occur early, on the third or fourth day; on the other, it may be retarded until the seventh or eighth day. In either case, if the subsequent course of the vaccination is typical, the vaccination may be regarded as satisfactory. When, however, the course is otherwise not typical, the presumption is strong that the vaccination is not good. Instead of pursuing the ordinary course the vesicles may appear upon an inflammatory base and abort in two or three days; or they may attain their maximum development in two or three days with turbid instead of clear contents and without an inflammatory areola. All such atypical vaccinations are to be regarded with suspicion. They are usually the result of the use of virus which is not active.

Complications.—Vaccination is frequently complicated by contamination with pus organisms, which produce violent inflammatory reaction. In such cases cellulitis follows and deep excavating ulcers with necrotic centers may form, accompanied by much pain and considerable systemic reaction. The cellulitis in these cases may vary from a small circumscribed area the size of a silver dollar to an extensive involvement of the part. This complication is not vaccinia but cellulitis from infection, and the presumption is strong against a successful vaccination in such cases. The scar which results from an unsatisfactory vaccination of this sort is a smooth or perhaps keloidal scar, such as follows other simple ulcerative processes.

It is possible for inoculable diseases to be transmitted by vaccination. The ones most to be guarded against are tuberculosis and syphilis. Syphilis can be perfectly guarded against by using bovine virus. The use of bovine virus probably does not exclude the possibility of inoculating tuberculosis, but the accident must be one of great rarity. If syphilis is inoculated in vaccination there results the development of a chancre at the site of inoculation, and, as the period of incubation of syphilis is longer than the entire period of vaccinia, the chancre appears after the vaccination has run its course.

It is not uncommon for pustular dermatitis to develop in connection with vaccination. The course of this complication is usually as follows: the vaccination lesion becomes infected with pyogenic organisms through lack of care; as a result of itching around the vaccination the patient scratches—the patient is usually a child—and inoculates other points; and so the disease is spread from point to point. In this way very extensive cases of pustular dermatitis occasionally develop. In rare instances generalized bullous eruptions occur due to local infection (bullous impetigos). While at times extensive, they are as easily cured, as a rule, as other impetigos.

There is another class of eruptions which occurs with vaccination as result of the general intoxication. These are most frequently toxic erythemas. In rare instances bullous eruptions have occurred which are undoubtedly due, not to local causes, but to profound constitutional disturbances. These latter are practically the only complications of a serious character which cannot be guarded against; they are of excessive rarity, and have usually occurred only in individuals who have been greatly reduced by dissipation or by unhygienic surroundings.

The following classification, adopted from Malcolm Morris, indicates the various dermatoses which may possibly follow vaccination:

1. Diseases due to the vaccine virus itself.	Local	Normal vaccination.	
		Dermatitis (areola).	
		Adenitis.	
		Generalized vaccinia.	
	Constitutional	Generalized vaccinia.	
		Miliaria.	
		Roseola.	Toxic Dermatoses.
		Papulo-vesicular dermatitis (Vaccinia lichen, Crocker).	
		Purpura.	
		Erythema multiforme.	
		Urticaria.	
2. Diseases due to mixed infection at the time of vaccination or later.	Local	Impetigo contagiosa.	
		Cellulitis.	
		Erysipelas.	
		Furunculosis.	
		Local gangrene.	
	Constitutional	Sepsis.	
		Tetanus.	
		Syphilis.	
		Tuberculosis.	
		Leprosy.	
3. Eruptions sometimes following vaccination without clear causal relations.	Eczema.		
	Psoriasis.		
	Bullous eruptions.		
	Acute pemphigus.		
	Dermatitis herpetiformis.		

The occurrence of such of these diseases as are due to infection with the ordinary pyogenic or with specific pathogenic organisms is self-explanatory. In vaccination one produces an abrasion of the skin in which any inoculable disease may find lodgment, just as it may in any other abrasion. These complications of vaccination can be guarded against by care in vaccinating and by the selection of well-prepared bovine virus. The severer complications from contamination of pyogenic organisms, like severe cellulitis and gangrenous ulcers, are very uncommon with careful vaccination at the present time. The toxic erythemas are usually trivial complications of vaccination. They are the result of the systemic intoxication which to a greater or less extent inevitably accompanies vaccination, and they can no more be guarded against and are no more to be dreaded than are the same sort of dermatoses as they occur from the use of antitoxin in diphtheria.

The dermatoses which cannot be guarded against and which are serious are the bullous eruptions, which seem to be produced either by a peculiar general infection or by a profound systemic disturbance of some other sort. I reported a few years ago a case of dermatitis herpetiformis which followed vaccination so closely as to leave no doubt of a causal relationship. More recently Bowen, Stelwagon, and Sequeira have reported similar cases which are to be regarded either as dermatitis herpetiformis, pemphigus, or bullous erythema multiforme. The relationship of vaccination to these conditions is obscure. In my case it seems probable that the predisposition to dermatitis herpetiformis already existed, and that the reaction of vaccination was simply the exciting factor. In another group of cases which have been reported by Howe, of Boston, it seems highly probable that the bullous dermatitis was due to the diffusion through the system of an infection which had been introduced at the time of vaccination, and that the cases are analogous to the cases of acute pemphigus occurring from inoculations in butchers which have been described by Pernet. All of these peculiar bullous eruptions are excessively rare, for with the innumerable vaccinations only a few have been observed. They have occurred chiefly in persons who are dissipated and much below par physically.

Any essential relationship of vaccination to eczema or psoriasis is extremely improbable. In a predisposed individual an outbreak of either of these dermatoses may be excited when the patient's resistance is reduced by vaccination, as it might be by any other constitutional disturbance.

VI.—VARICELLA

SYNONYM: *Chicken Pox.*

Incubation.—The incubation period of varicella is variable. It is, as a rule, longer than that of smallpox, measles, or scarlatina. It is usually about fourteen to seventeen days (Welch and Schamberg).

Invasion.—The prodromal symptoms of chicken pox are of short duration and intensity. They consist in rise of temperature, usually not above 100° F., with the accompanying symptoms of a febrile disturbance, chilliness, anorexia, nausea, slight headache, and backache. High fever, vomiting, and great prostration, such as characterize smallpox, hardly ever occur. It is quite frequent for no symptoms to be noticed until the eruption appears. When the disease affects adults, as it occasionally does, the prodromal symptoms are more apt to occur and may precede the eruption two or three days. Rarely a prodromal erythema occurs with varicella as it does with smallpox.[1]

Eruption.—The eruption appears usually upon the back, sometimes upon the face, and rapidly spreads to other parts. It is most profuse upon the trunk, especially upon the back, and is least abundant upon the extremities below the elbows and knees. The number of lesions upon the hands and feet is usually small, but lesions may occur upon the palms and soles. The scalp and face are also regularly involved. Its distribution may be said in a general way to be least abundant upon those surfaces where smallpox is most abundant—the hands and feet and face—but it does not entirely avoid any of these parts. The extent of the eruption is variable. There may be only a dozen lesions over the entire body. On the other hand, they may be abundant and generally distributed, amounting rarely to two or three thousand.

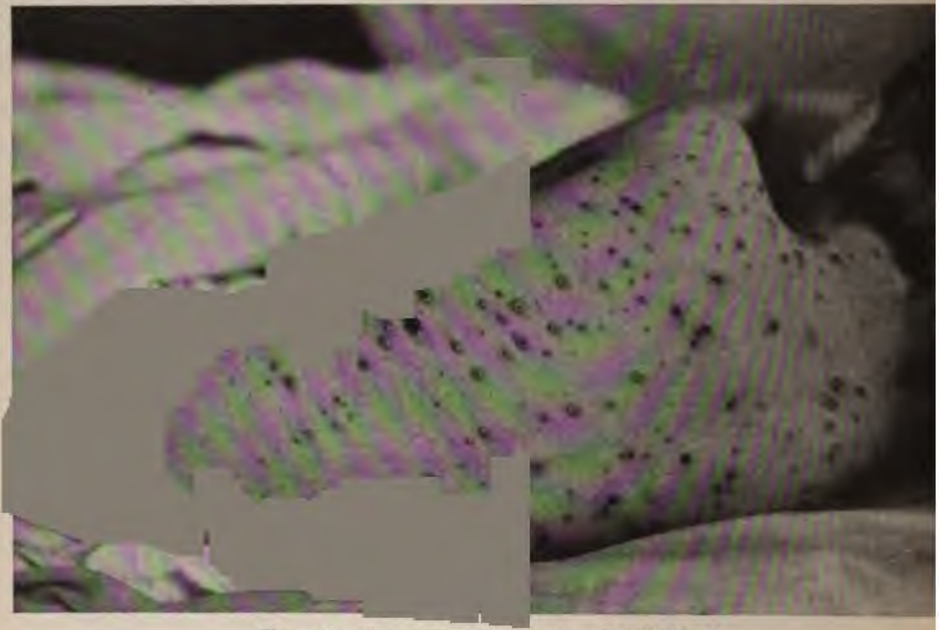

FIG. 42.—VARICELLA. (Schamberg's collection.)

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[1] Anthony (*Jour. Cutan. Dis.*, Feb., 1906) has described a prodromal erythema lasting a few hours which he has observed in two cases of chicken pox and which he believes is exactly analogous to the prodromal erythema of variola.

The lesions of chicken pox appear first as red macules which almost immediately become papular, and in four to six hours develop into clear vesicles upon a reddened base. The vesicles quickly become opalescent, and within thirty-six to forty-eight hours become pustular and either rupture or desiccate, being converted into crusts by the third or fourth day. These quickly exfoliate, leaving temporary stains, and, after a few of the lesions, scars. The eruption of varicella does not occur all at once, but appears in crops on successive days, there being ordinarily two or three crops, so that about the third day one can find lesions in all stages of evolution. The entire course of the eruption may extend over six to eight days. The lesions of varicella vary greatly in size, from a pinhead to a pea. They are superficial, and are smaller as a rule than the lesions of smallpox and without the markedly inflammatory base of the smallpox pustule. The vesicles are not umbilicated, and when fully developed the pustules are not the prominent striking lesions that smallpox pustules are. When a varicella pustule begins to desiccate, the crust at the center settles down and forms a depressed center with a vesicular ring around it. This is a pseudo-umbilication which is similar to the same sort of umbilication occurring in the smallpox pustule. It is not to be confused with the true umbilication which occurs in the smallpox vesicle. Occasionally there will be a very slight puckering or conical umbilication of a chicken-pox vesicle which is formed around a hair. This differs both in appearance and essentially from the true umbilication of smallpox, and should not lead to confusion.

Occasionally in delicate children, especially in the tuberculous, bullous eruptions develop in varicella, and Jonathan Hutchinson has called attention to gangrenous ulcers in the skin which occur as a sequel of varicella. This occurs especially in tuberculous children. It is not a true modification of the eruption of varicella, but a complication due to mixed infection in patients of low resistance, and has been observed repeatedly in other dermatoses (Dermatitis Gangrenosa Infantum).

Varicella has to be diagnosticated from mild variola. The points of difference are the more superficial character of the lesions, the lack of umbilication of the vesicles, the preference of the eruption for the trunk, and the shorter course of the lesions, the varicella lesion becoming a crust in about four days, the variola lesion in ten to twelve days. More important still is the difference in clinical history, the short, mild, and rather characterless prodromal illness of varicella differing strikingly from the regular and severe period of variola.

The points of contrast in the common features of the different exanthemata are shown in the following table:

	SCARLET FEVER.	MEASLES.	GERMAN MEASLES.	SMALLPOX.	CHICKEN POX.	VACCINIA.
Chronology.						
Incubation..........	3 to 7 days.	10 to 12 days.	5 to 20 days.	10 to 12 days.	14 to 17 days.	2 to 3 days.
Invasion............	1 to 2 days.	3 to 4 days.	Fewhours to 1 or 2 days.	Until the 3d to 5th day.	24 hours.	Small papules 3d or 4th day. Vesicles 5th to 6th day; maximum on 8th day. Vesicles begin to desiccate 10th to 11th or 12th day. Crust forms 14th to 15th day, which separates 21st to 25th day from date of vaccination.
Invasion............	Onset sudden with high temperature and angina.	Not so sharp as in scarlet fever. Rapid rise of temperature on 1st day, 103° to 104° F., with remission of about 2° until eruption appears. Catarrhal symptoms marked.	Short, with very slight mild febrile symptoms.	Onset sudden with chill, high fever, intense headache and backache, and severe constitutional symptoms.	Short and symptoms mild.	
Eruption............	Appears 2d to 3d day. Fully developed in 24 hours. Fades 5th to 7th day.	Appears 4th day. Complete in 24 to 36 hours. Fades on 6th to 8th day.	Appears 1st to 2d day.	4th to 12th day. Desiccation begins about 10th day.	Appears 2d to 3d day.	
Desquamation.......	2 to 4 weeks; ending from 4th to 6th week of disease. Very abundant.	Begins on 7th to 8th day. Complete in 12 to 15 days.	Begins 3d to 4th day. Ends 6th to 7th day.	Begins 12th day. Ends 21st to 28th day.	7th to 8th day.	
Character of eruption............	Bright red small macules.	Large dark red maculopapules. Often arranged in crescentic or circinate figures.	Light red macules; size pinhead to pea or larger, may simulate either scarlet fever or measles.	Papules on 4th day. Vesicles on 5th to 6th day. Pustules 8th day. Desiccation begins 10th to 12th day. Desiccation well advanced 14th to 15th day.	Appears in 2 or 3 crops in the course of 2 or 3 days. First superficial red papules, in a few hours becoming vesicles, in 36 to 48 hours pustules which quickly begin to dry up. A single lesion runs its course in 4 days. Entire eruption runs a course of 6 or 7 days.	
Points of first appearance.........	Side of neck and front of chest.	Cheeks and forehead.	Face and upper part of trunk.	Face, hands, and feet.	Back and chest.	
Distribution and extent of eruption..	Over the entire body except around the nose and mouth. Entire face may escape. Lesions so abundant as to produce diffuse scarlet color of the entire surface.	Most abundant upon face, neck, upper part of trunk, and upper extremities. Very rarely abundant enough to cause universal diffuse redness.	Face, neck, trunk, and extremities; discrete lesions; not in crescentic arrangement and usually not abundant.	In mildest cases few lesions on face, hands, and feet. In severest cases confluent on head and extremities, very abundant over rest of body.	Varies from a dozen or so lesions on the trunk to many hundreds which are abundant on trunk with few scattered lesions elsewhere. Never confluent.	

VII.—"THE FOURTH DISEASE"[1]

Dukes[2] has undertaken to describe, under the name "The Fourth Disease," an exanthem which he thinks has been confused with German measles. As Griffith[3] and Marsden[4] have pointed out, however, there is nothing to show that the cases which he has described are not aberrant forms of other exanthemata, especially German measles and scarlet fever.

VIII.—"THE FIFTH DISEASE"

Watson[5] has described from seventy-five cases a disease which he believes differs from the other exanthemata. The distinction remains to be established.

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[1] Curtis, "An Epidemic of the Fourth Disease," *Jour. A.M.A.*, August 30, 1902.

[2] *Lancet*, July 14, 1900.

[3] *Philadelphia Med. Jour.*, April 12, 1902.

[4] *Lancet*, August 16, 1902.

[5] *Boston M. and S. Jour.*, June 24, 1909.

SECTION III

ANGIONEUROTIC DERMATOSES

Beginning with symptomatic erythema or roseola considered, there is a number of important diseases whic be grouped together on account of the similarity of t processes which produce them.

All of these diseases are characterized by lesions w localized vasomotor or trophic disturbances in the skin. this there is congestion and an inflammatory exudate of intensity. In hyperæmic or simple erythema this amo superficial congestion with just the first traces of inflam In exudative erythema there is a definite inflammatory e formation of papular or vesicular lesions. In purpura, the dominant characteristic of the inflammatory process an the condition both anatomically and clinically. In urticar is still essentially an inflammatory process, but with the blood serum as the chief factor, the other inflammatory fea less marked; so that there are produced papular lesions of an character. In herpes, herpes zoster, pemphigus, and dermatitis formis the inflammatory process is characterized by a violent o of serum which results in the rapid formation of tense vesicles

In all of these conditions the process is a disturbance of the tone.[1] In the conditions last enumerated, from herpes to pemphig processes are, perhaps, less of a vasomotor character than distur of all of the factors controlling the nutrition of the skin—violent tr disturbances.

These various conditions present common causal features. All them may be regarded as conditions which are produced by disturban of the nervous mechanism which controls the vascular tone of the ski or of the mechanism which controls the nutrition of the skin. These disturbances may be either of the central or of the peripheral mechanism. The causes which underlie one or another of these conditions may be classified as follows:

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[1] Martin Fischer (*Jour. A.M.A.*, 1908, vol. li, p. 830) has shown that the mani festations of œdema and of many of the changes of inflammation and of hæmorrhag in the skin may be explained by varying affinities of the tissue colloids for water o blood plasma and by the reactions of colloids of different densities upon each othe It is possible that with the increase of knowledge in this extremely complex subje we will find that less importance than is now attributed to it belongs to the vas motor nervous control, and much more to the reactions of the tissues and fluids.

(1) Emotional and other forms of reflex irritation.

(2) Organic nervous diseases.

(3) Toxic disturbances. These may be of various sorts. They may be due to foods or drugs, to toxins produced by bacteria either within or without the body, or to toxic products resulting from metabolic disturbances.

(4) External sources of irritation, which probably act, in urticaria for example, by temporarily disturbing the peripheral nervous mechanism which controls the tone of the cutaneous blood vessels of the injured point.

A certain factor of personal susceptibility is frequently apparent when these diseases occur. Many of them are most frequent in childhood when nervous stability is least firm and metabolic activity greatest.

Erythema multiforme, urticaria, and purpura are diseases of analogous character. They occur from similar causes, and the characteristic lesions of the different diseases not infrequently occur together, producing mixed types of eruptions. Thus with the purpuras we may have urticarial lesions, and the same happens with erythema multiforme. The inference, therefore, is fair that the diseases are essentially related, although in their typical forms and in their extreme types they may be very far apart.

As Osler in particular has shown, what he calls the erythema multiforme group of skin diseases may be the manifestation of profound metabolic or bacterial disturbances, although fortunately they are usually manifestations of ephemeral disturbances of this sort.

THE EXUDATIVE ERYTHEMAS

I.—ERYTHEMA MULTIFORME[1]

SYNONYM: *Erythema exudativum multiforme.*

Erythema multiforme is a disease of the skin characterized by the development of subacute papular or vesicular lesions which are symmetrically distributed and show a marked predilection for the dorsal surfaces of the extremities below the knees and elbows.

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[1] Duhring, *Archiv*, 1896, vol. xxxv, pp. 211 and 323 (complete review).—Osler, *Amer. Jour. Med. Sci.*, January, 1904, and December, 1895.—Schamberg, *Jour. Cutan. Dis.*, 1904, p. 461.—Galloway and MacLeod, *Brit. Jour. Derm.*, March, 1903. —Pond, *Med. News*, 1904, p. 506.

It manifests itself in eruptions which vary greatly in because of variations in size, character, arrangement, and of the lesions. All of the types of the eruption, however, ha in common which indicate the essential unity of the disease.

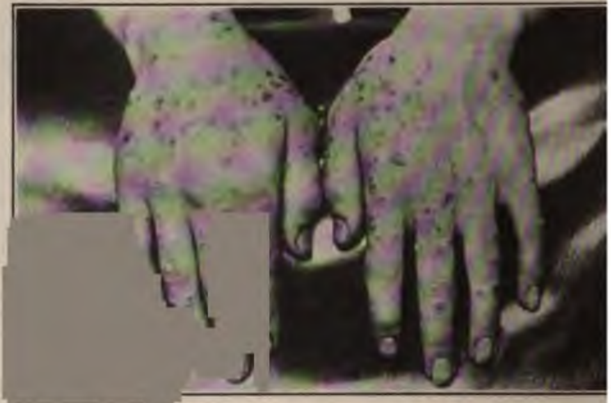

FIG. 43.—ERYTHEMA MULTIFORME OF HANDS AND WRISTS
(Author's collection.)

According to the form which the eruption takes, eryth forme is described as:

Erythema papulatum vel tuberculatum,
Erythema vesiculosum vel bullosum,
Erythema circinatum et gyratum,
Erythema marginatum,
Erythema iris vel Herpes iris.

Erythema multiforme constitutes about one half to one cutaneous diseases. It is not a very common disease, nor is Erythema papulatum is the commonest form, erythema marg erythema iris and herpes iris are rare, the last the rarest.

Symptomatology.—The elementary lesions of eryth forme are inflammatory papules or tubercles of a pinkish t color, slightly elevated and rounded rather than pointed, a in size from a pinhead to a pea—larger than the same lesion dermatitis or eczema. Macroscopically they present the evid inflammatory process of mild intensity. They enlarge aft pearance, reaching their full size within twenty-four hours. full development their pinkish color changes to purplish.

In all forms of erythema multiforme occasional discrete, tubercular lesions occur like those of the papular and tub of the disease. In any of these forms the inflammatory pro intense enough at certain points to produce vesicles or bulla

les or tubercles, so that the appearance of vesicular lesions may com-
te the picture (*Erythema vesiculosum vel bullosum*). Hæmorrhage
occasionally takes place into the lesions and in all forms of the
se petechiæ and urticarial wheals may at times be seen. In the
est form of erythema multiforme (*Erythema papulatum, tubercu-*
, vel tuberosum) the lesions end their evolution with these inflam-
ry papules or tubercles. In such cases the lesions may remain dis-
with only occasional small groups coalescing into patches. The
se then presents the appearance of a scattered papular or tubercular
ion separated by areas of normal skin. More frequently, however,
sions occur closely aggregated in patches, and as they enlarge they
elevated plateaux of mottled pink or purplish red which are mani-
produced by the coalescence of papular lesions. Papular and tuber-

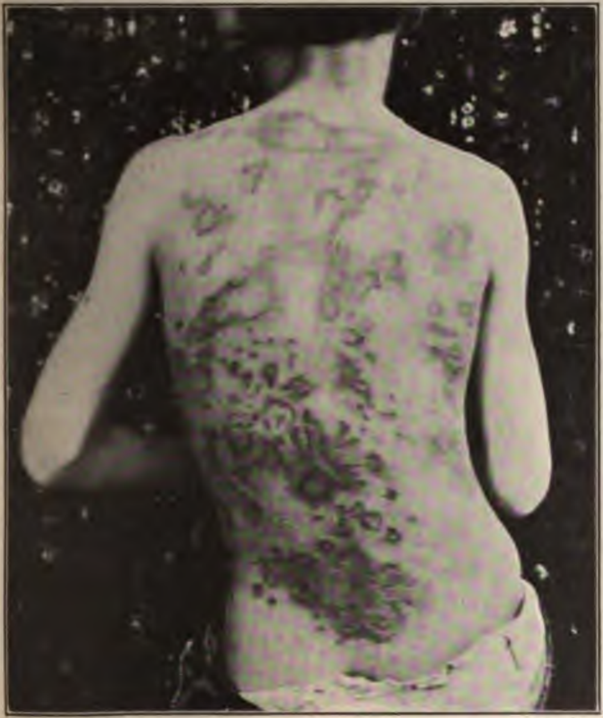

Fig. 44.—Erythema Circinatum. (Author's collection.)

r erythema appears symmetrically upon the backs of the hands and
, spreading more or less to adjacent parts. On the dorsal surfaces of
hands it is very likely to form patches, with discrete lesions upon
acent parts and upon the face.

In another form (*Erythema circinatum vel annulare*) the lesions appear as pink or purplish flat papules, perhaps as large as a ten-cent piece, which spread at the periphery while subsiding at the center. In this way ringed lesions are formed with a border which represents the active process while the center is undergoing involution. The border of such lesions will be red or purplish, slightly elevated, rather sharply defined on its outer edge but fading away more gradually upon the inner edge. Within this reddish or purplish ring the skin is a fainter shade of red or purple or brownish. These rings may enlarge to the size of one and a half inches or more in diameter. When the rings are close together they come in contact as they spread, and, following the rule of coalescing ringed lesions, in the lines of contact the active process subsides and the rings are broken. It can readily be seen that by the confluence of numerous rings of this sort peculiar gyrate figures may be formed by the eruption (*Erythema gyratum*).

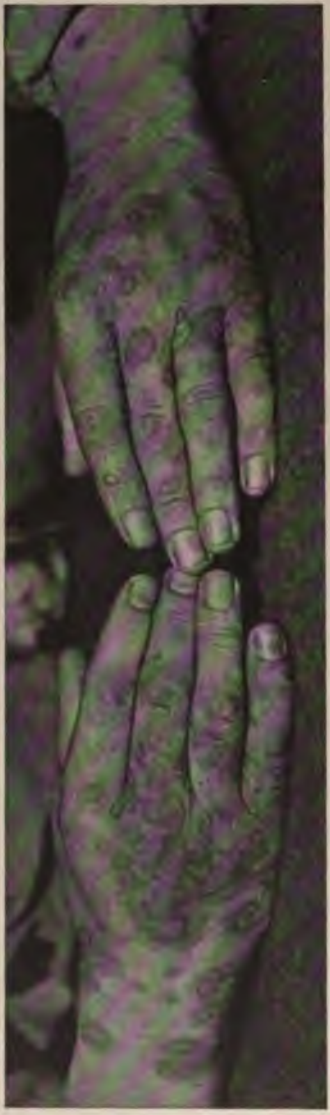

FIG. 45.—ERYTHEMA MULTIFORME BULLOSUM, IRIS-FORM. Lesions also on palm. (Author's collection.)

In another form, closely similar to the above, the disease begins either as large, flat papules or as erythematous patches which spread rapidly at the border while they subside at the center. This form differs from the preceding in that the lesions spread much more widely and the border is less elevated. By the extension of the lesions very large circinate or gyrate figures are formed with a chamois-colored center and with brownish-red borders very slightly elevated (*Erythema marginatum*).

At times the rings of erythema multiforme show distinct zones of color. This phenomenon may be due simply to variation in the stage of subsidence of the process; a brownish center, outside of this a purplish zone, and outside of this a zone of a brighter shade of red. It is more frequently, however, due to the development of a central papule; then a papular ring develops around this and so on, thus producing compound lesions around a common center. The papular lesions so produced constitute *Erythema iris.*

Herpes Iris.—The most striking form of erythema multiforme is that in which the lesions occur in concentric rings that become either vesicular or bullous. This differs from erythema iris, referred to above, in which the iris formation is due to concentric zones of different color but without vesicle formation. In herpes iris the lesion begins as a ring of erythema multiforme which, after progressing to a certain extent, develops bulla at its center. Around this there then appears a circle of vesicles, and this process may continue with the formation of several concentric circles. The lesions thus formed, when completed, consist of central bullæ surrounded by one or more concentric circles of vesicles. Usually there are not more than two or three rings, but as many as seven can be counted in the case pictured in Wilson's *Atlas.* The contents of the vesicles are opalescent, and the skin underlying these lesions is of a purplish-red color. This form usually occurs in individuals who are physically below normal.

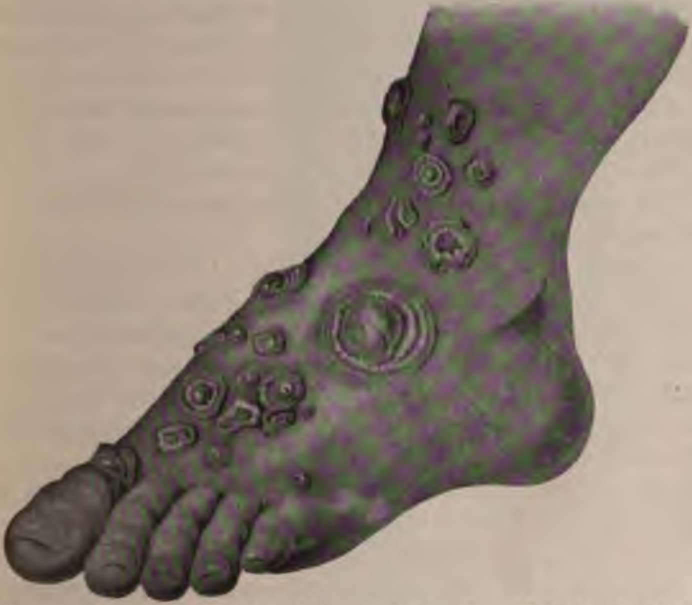

FIG. 46.—HERPES IRIS. (Photographic reproduction from Hebra's colored plate.)

The eruption of erythema multiforme shows a marked tendency to symmetry. The symmetry may not be exact, but the eruption is practically always bilateral and involves corresponding surfaces. The parts of predilection of the eruption are the extremities below the elbows and knees—especially the backs of the hands and next most frequently the feet—the face, and to a certain extent the trunk. In the case illustrated in Fig. 44 (p. 211) the disease was confined to the trunk, chiefly to the back, and Jamieson has seen it on the face alone, but in almost all cases it is most abundant upon the extremities, and rarely fails to involve the backs of the hands. Herpes iris usually occurs on the hands and feet alone, except for satellite lesions which spread upon the wrists and ankles. The bullous and vesicular lesions also usually occur upon these parts alone, although the bullous lesions may occur upon the face. The papular and circinate forms tend to a wider distribution, and the lesions occur frequently on the face and, to a certain extent, over the trunk. The large circinate forms are apt to occur more abundantly upon the trunk than the other forms.

The extent of the eruption is variable. It may consist of only a few lesions upon the backs of the hands or hands and feet. More frequently it extends up on the wrists and around on the palmar surfaces, and as a rule a greater or less number of lesions are found upon the extremities below the elbows and knees. The eruption may be general and abundant, involving in some cases not only the skin but also the mucous membranes of the eyes and mouth.

The appearance of the eruption in erythema multiforme is usually sudden. The first lesions to appear reach their full development in about twenty-four hours, then may remain unchanged for two or three to five or six days, and then subside, finally disappearing in the course of ten to fourteen days, leaving only brown stains behind. As a rule these stains follow the disease and persist for some time. Herpes iris occasionally becomes purulent and the lesions dry into crusts. In a case of Crocker's scars formed, but this is very unusual. Ordinarily the lesions do not become purulent; the serum is absorbed or the vesicles rupture, and the loosened epidermis exfoliates. The entire eruption does not as a rule appear at one time, but crops of lesions appear from day to day, it may be for a week or ten days, so that after a week lesions in all stages of their course may be seen in a given case, and the entire duration of an attack may be prolonged for three to four weeks or even longer. The average duration of an attack is two to three weeks.

The eruption of erythema multiforme thus runs a definite course and is self-limiting. The disease, however, shows a tendency to recur, and cases have been reported by Colcott Fox and others in which the relapses have succeeded one another so rapidly that the disease persisted for months or indefinitely (Erythema perstans).[1] Herpes iris tends to frequent recurrences, perhaps three or four times a year.

Ordinarily the subjective symptoms accompanying erythema multiforme are trivial. There may be slight feeling of tension and burning. Itching, if present, is mild. In some cases these symptoms are more acute, but the mildness of the itching in comparison with other inflammatory dermatoses is a characteristic of the disease, unless there are urticarial wheals present. When itching occurs in the vesicular and bullous forms the lesions may exceptionally be painful.

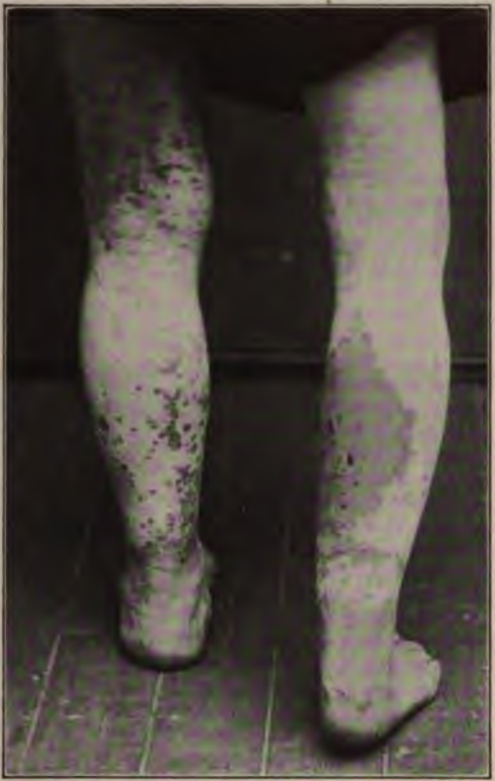
FIG. 47.—ERYTHEMA MULTIFORME PERSTANS. (Winfield's collection.)

Constitutional disturbances are usually slight or absent. Patients may make no complaint of feeling out of health, and frequently no disturbance of health can be discovered. At times there is more or less systemic disturbance of the sort which accompanies mild forms of toxæmia. There is slight rise of temperature with the usual accompanying symptoms, and frequently there are articular pains. In very rare cases there is an acute febrile disturbance with temperature of 103° or 104° F., and severe pains in the joints resembling acute articular rheumatism. The constitutional disturbances, when they occur, may precede by a variable period of a few hours to several days the appearance of the eruption, and are likely to continue for some time after its appearance or until the eruption begins to subside. Jarisch has noticed enlargement of the glands, especially of the cervical glands, in some cases. As Osler especially has shown, the different forms of erythema multiforme may be manifestations of severe, perhaps fatal, constitutional disturbances of metabolic or toxic character with diverse visceral lesions. These cases do not correspond to the accepted picture of erythema multiforme in which the systemic disturbance is practically of no importance; they are, however, of interest as throwing light upon the toxic processes which underlie at least a large proportion of the cases.

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[1] The condition known as Erythema Perstans is sufficiently characteristic to form a fairly definite clinical entity. It is a form of erythema showing superficial inflammatory changes, with an abundant and symmetrical eruption which is permanent. Numerous cases have been described since Colcott Fox (*International Atlas*, V, 1901) called attention to the disease. Wende (*Jour. Cutan. Dis.*, June, 1906) has recently reported two cases; his article gives histology and a review and bibliography of previous cases.

Etiology.—Erythema multiforme is an angioneurotic disturbance produced by some systemic impression upon the vasomotor apparatus controlling the blood vessels of the skin. It may possibly be of reflex nervous origin, but there seems every reason to believe that in the vast majority of cases it is produced by some toxic substances in the system causing a temporary disturbance of the vasomotor regulation.

Personal susceptibility is apparently a factor in many cases. In such individuals the disease may be excited by local irritation. Crocker believes that he has seen it occur on the extremities from exposure to cold, to sunlight, and to sea winds in predisposed individuals. Kaposi and Lewin think that urethral irritation, and Hebra and Pick that uterine disturbances, may produce it. But such causes are uncommon, and as a rule it is undoubtedly produced by toxic disturbances. Duhring and Stelwagon believe it is frequently excited by foods; such foods as cause urticaria, especially those containing products of decomposition. Stelwagon has observed it follow the use of potassium iodide, copaiba, and the coal-tar products. There is every reason to believe that intestinal toxins play an important part in its production. I have seen it alternate with eczema on the backs of hands of a woman subject to an autointoxication eczema of the hands. Its association with gout and chronic rheumatism in patients who are affected by the presence of products of disordered metabolism is a common occurrence. In most cases it is probably the result of some constitutional infection, the character of this varying in different cases. Cocci have been found in the blood by Cordua and Luzato; bacilli and spores by Manssurrow; various bacteria which are suggested as pathogenic have been described by Simon, Leloir, Finger, and others; and many writers believe that it is an acute specific disease. It is not likely that this is true. It seems more probable that its eruptions may be produced by various infections and other toxic disturbances.

It occurs most frequently in spring and fall, and in damp, changeable weather. It is most frequent during adolescence and early adult life and is slightly more frequent in females. The urinary findings are indefinite.

Pathology.—The disease is an inflammatory process showing all of the features of inflammation. The inflammatory exudate may be only sufficient to form solid papular lesions, or it may uplift the epidermis with the formation of cavities filled with serum. As in inflammatory processes generally, there is always some diapedesis of red cells, the disintegration of which producês the brown staining which persists after the lesions disappear. In certain lesions free extravasation of blood occurs into the tissues with the formation of petechiæ, and rarely extravasation of blood is a feature of the eruption. In a lesion of tubercular erythema examined by Crocker the changes were essentially those of a moderate inflammation of the upper part of the corium. In lesions of herpes iris examined by Pardee the findings were practically the same, an exudative inflammation in the upper part of the corium with the formation of bullæ.

Diagnosis.—The picture presented by erythema multiforme in its different types is characteristic and not readily confused with other conditions. The symmetry of distribution, the predilection for the hands and feet, the absence of itching, the mildness of the constitutional symptoms, and the self-limiting course, form a peculiar symptom complex. Papular erythema multiforme is distinguished from papular eczema by the larger size of the lesions, the absence of any pinpoint vesicles, the lack of itching, the peculiarity of distribution, and its self-limiting course. Papular erythema multiforme may be confused with German measles. The lesions, however, are larger and more distinctly papular. Their color tends to purple, the catarrh and angina are lacking, as are usually the enlargement of the auricular and postcervical glands and the articular pains. The course of erythema multiforme is longer. Erythema multiforme is distinguished from urticaria, unless the two conditions occur together, by the symmetry of its distribution, its definite course, and by the absence of the evanescent character of urticarial wheals and of the itching which is a part of urticaria. The iris forms of erythema multiforme are scarcely to be confused with any other dermatosis. The bullous forms might possibly be confused with pemphigus or dermatitis herpetiformis. The symmetry of distribution, the characteristic localization, and the more infiltrated inflammatory condition underlying the lesions would sharply distinguish typical cases of bullous and vesicular erythema multiforme. When, however, we come to the rare, very severe cases of erythema multiforme with widely distributed bullous and vesicular lesions, associated perhaps with papular and urticarial lesions and perhaps involving the mucous membranes, the diagnosis becomes extremely difficult. Indeed, it may be impossible, for there occur transitional cases whose nosological position is uncertain. These cases are of especial interest as throwing light upon the relationship of all this group of angioneurotic disturbances, and upon the similarity of the pathological processes which underlie them all.

Prognosis.—The different types of erythema multiforme, as they occur in the vast majority of cases without considerable systemic disturbances, disappear in two to four weeks, leaving stains which disappear in the course of a few weeks more. Very rarely, in the pustular bullous forms, scarring may occur. The disease is apt to recur, most probably at the same time the next year. Herpes iris tends to recur twice or three times during the course of the year, and this may be repeated for several years. In the very rare cases associated with visceral complications the prognosis is that of the complications.

Treatment.—The internal treatment of cases of erythema multiforme as ordinarily seen is concerned only with correcting disturbances of digestion, improving elimination, and meeting such other general indications as the cases present. It is usually desirable to clean out the intestines with a saline or mercurial cathartic, and to repeat this procedure at intervals of a few days. Intestinal disturbances are especially to be looked out for, as are gout and rheumatism. These are treated in the usual way. In the persistently relapsing cases especial attention has to be given to autointoxication from the intestinal tract, and to means for overcoming it. Various remedies have their special advocates in certain forms of the disease, such as the salicylates and salicin, potassium iodide, quinine, and ergot.[1] When the disease occurs in individuals who are enfeebled, tonics are strongly indicated, especially arsenic, strychnine, and quinine.

Little is demanded in the way of local treatment. If there is itching or burning an antipruritic application is indicated, such as equal parts alcohol and water, or cologne water and water, or vinegar and water, or calamine lotion, to any one of which it is desirable to add one half to one per cent carbolic acid. When bullæ form they should be ruptured, if they are tense and accompanied by disagreeable sensations, and the surfaces should be protected against infection by dressings of absorbent cotton, moist dressings of boric-acid solution, or a bland ointment, like boric-acid vaseline. If bullous lesions are not uncomfortable it is better to leave them unruptured and to protect them by dry dressings of cotton.

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[1] Granville McGowan strongly advises ten-drop doses of 1:1000 solution of adrenalin chloride every two hours in erythema multiforme and angioneurotic dermatoses (*Jour. Cut. Dis.*, 1905).

II.—ERYTHEMA NODOSUM[1]

SYNONYM: *Dermatitis contusiformis.*

Erythema nodosum is an acute inflammatory disease of the skin of systemic origin, characterized by the formation, especially over the tibiæ, of multiple, painful nodes which disappear spontaneously in the course of ten days to two weeks without ulceration.

The picture which erythema nodosum presents differs strikingly from that of erythema multiforme. However, its distribution, its course, its appearance in successive crops, and its toxic origin, all suggest its relationship to erythema multiforme. But more important still is the well-established observation that lesions of erythema multiforme sometimes occur with erythema nodosum.

Symptomatology.—The lesions of erythema nodosum are inflammatory nodes situated deeply in the skin. Before their appearance there are tender points in the skin over the tibiæ, and in the course of a few hours to a day or two there develop roundish or oval swellings with their long diameter parallel to the axis of the leg. These swellings are at first bright red and are usually elevated, although most of their bulk is below the surface of the skin. Their outline is ill-defined and their borders merge gradually into the surrounding tissues. They look, in fact, exactly like circumscribed areas of cellulitis from traumatism or infection. Their size varies from a hazelnut to a hen's egg. They are very tender and painful on pressure. They attain their complete development in from a few hours to a day; then they gradually subside, undergoing in their involution the changes which are seen in a bruise. At first bright red, tense, and glistening, they become purplish or violaceous, then bluish and greenish yellow, and finally disappear, leaving brown stains. As they begin to undergo involution they become soft and boggy as though suppuration were taking place, but this never occurs, and the mistake must not be made of incising them to evacuate supposed pus.

In number the lesions vary from one or two on either leg to a dozen nodules or (rarely) more. They are usually confined to the skin over the anterior surfaces of the tibiæ; but they may occur on the dorsal surfaces of the feet and the extensor surfaces of the forearms and the backs of the hands. Crocker has seen them over the scapulæ and the condyles of the humerus and on the thighs. Duhring noted their occurrence on the mucous membranes, and Crocker has seen a split-pea-sized nodule in the sclerotic of the right eye in connection with lesions on the legs. The individual lesions run their course in from one to two weeks. The eruption, however, does not all appear at one time; usually two or three lesions appear at a time and new crops appear from day to day, the whole eruption persisting for two or three weeks; or occasional crops may continue to appear so that the disease persists for four or five weeks or longer.

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[1] S. Mackenzie, *London Clin. Soc. Trans.*, 1886, vol. xix, p. 215 (analysis of 108 cases).—Harrison, *Brit. Jour. Derm.*, 1900, p. 250 (analysis of 80 cases).—Abt, *Jour. Amer. Med. Assoc.*, November 12, 1904 (Literature).

The disease is usually ushered in by slight febrile disturbances commonly associated with articular pains and perhaps swellings. These symptoms, except the articular pains, may be so mild as to escape notice, but occasionally there is an acute febrile disturbance which suggests beginning articular rheumatism. The temperature occasionally rises three or four degrees. The constitutional symptoms as a rule precede the eruption from a few hours to two or three days, and are likely to continue as long as new lesions appear.

Etiology.—Erythema nodosum is a disease of toxic origin. In rare cases it has been found to follow the administration of drugs, such as the iodides and antipyrin. In the vast majority of cases it is an accompaniment or a sequel of an infectious process, and it is most frequently associated with articular rheumatism. So close is this relationship that it may be regarded in many cases as a manifestation of rheumatism. As Stephen McKenzie has shown, it is frequently associated with definite rheumatic symptoms, such as articular pains, sore throat, and acid sweatings, and it may be complicated by endocarditis without the existence of articular lesions. In rare cases it has been seen to follow measles, diphtheria, and syphilis; more frequently it has been found in association with anæmia and malaria. Some cases have apparently been definitely traced to living under unhygienic surroundings with bad food and exposure to sewer gas. And epidemics of it have been observed among soldiers and in institutions and in persons who have been suddenly transferred to unfavorable surroundings. The occurrence of these epidemics seems to be explained either by some ill-defined intoxication as result of unsanitary conditions, or by some common infection. That there is any specific form of infection which accounts for all cases there is little reason to believe. The statistics of Stephen McKenzie strongly indicate that the commonest cause of the condition is rheumatic infection.

Erythema nodosum is uncommon, but not exceedingly rare. The disease occurs from three to five times as frequently in females as in males. It is most frequent between ten and thirty years of age. In McKenzie's 108 cases, 14 were under ten, 69 between ten and thirty, 15 from thirty to forty, and 10 over forty. According to Crocker it is not more frequent at one season than another, but in my experience it occurs especially in the changeable weather of spring and fall.

The disease is an intense inflammatory process which involves the corium and subcutaneous tissues, but not the epidermis.

Diagnosis.—The essential feature of erythema nodosum is the development in connection with a moderate febrile disturbance of multiple, painful nodes in the skin over the tibias, which become boggy but never suppurate, and which undergo the involution of a bruise. The only lesions they are likely to be confused with are syphilitic nodes and areas of cellulitis. Periosteal gummata at first are not red and are without the evidence of an acute inflammatory process, they develop much more slowly, they break down, and they are promptly affected by potassium iodide which does not affect erythema nodosum. Syphilitic nodes are not likely to be multiple or symmetrical unless they occur in the early course of syphilis, which would only be in exaggerated cases where other evidences of syphilis would be sure to be present. In congenital syphilis, where they may be numerous, they would be associated with other characteristic symptoms of inherited syphilis. Nodes over the tibiæ occurring in late syphilis bear little resemblance to erythema nodosum. The nodes are not multiple nor symmetrical, they develop more slowly, are not red at first, and they tend to gummatous degeneration. Areas of cellulitis are more diffuse and usually larger, they are rarely multiple or symmetrical, and they are apt to suppurate.

The differences from erythema induratum are considered under that disease.

Prognosis.—The disease runs its course in two to three weeks. It is not likely to recur, although recurrences have been noted. The possibility of endocarditis and permanent cardiac damage should be borne in mind.

Treatment.—Rest should be obtained to as great an extent as possible, and confinement to bed is desirable but not usually practicable. When the patients cannot be put to bed the best substitute is to bandage the legs firmly over a thin layer of absorbent cotton. Internal treatment consists in meeting general indications. The salicylates are most frequently useful, and after them quinine. The bowels should be kept open with salines or mercurials, and the diet should be bland and in febrile cases liquid. For the pain in the lesions agreeable applications are lead and opium wash in the strength of one half dram of solution of subacetate of lead and tincture of opium to the ounce of water, or ten per cent ichthyol ointment, perhaps with the addition of five to ten per cent of menthol. Jamieson advises painting the nodes with flexible collodion, to which might well be added ten per cent of ichthyol.

III.—ERYTHEMA ELEVATUM DIUTINUM[1]

Crocker and Williams have suggested the name erythema elevatum diutinum (persistent elevated erythema) for an eruption of sharply circumscribed elevated nodules or flat plaques of a reddish or purplish color which are associated with a gouty or rheumatic history and show little if any disposition to disappear. They suggest as probably of essentially the same character, although showing varying features, similar cases which have been reported by Middleton, Bury, Hutchinson, and others.

The condition is excessively rare, less than a dozen cases having been described.

The primary lesions are dense, pea- to bean-sized convex nodules, at first pink and later becoming purplish, or purplish from the beginning. They may remain as discrete nodules or develop into nodular tumors (Soemmering's case), but they tend to coalesce into purplish flattened plaques, either with or without a nodular surface. These plaques are sharply circumscribed, have an abruptly rising border, are very distinctly elevated, and are of dense consistence. In Hutchinson's cases the lesions were less firm and were œdematous, and could be made to disappear in part by continuous pressure. In a general way they suggest sarcomatous nodules and patches in the skin. In some of the cases there have been only a few lesions; in most instances they have been somewhat numerous. The distribution apparently follows no regular arrangement. In the cases of Bury, Crocker, and Williams the lesions showed no definite distribution. In most of the cases they were situated on the extensor surfaces of the limbs and over the elbows, the knees, and the phalanges of the hands and feet. They also occurred on the palms and soles, the buttocks, and the ears. In Hutchinson's cases they showed a tendency to appear at sites of pressure, and had no special predilection for the extensor surfaces of the joints. One of the most characteristic features is the persistence of the lesions. In most of the cases described the lesions have shown no tendency whatever to disappear, and in Hutchinson's cases they have tended to spread widely. In Crocker's cases and in the doubtful cases of Stelwagon the lesions have shown no tendency to spread over large areas, and involution in some of the lesions has ultimately taken place.

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[1] Cases which are widely different in themselves but which perhaps belong in the same group have been described by Soemmering (*Hutchinson's Archives*, vol. ii, 1891) and Gilchrist (*Trans. of the Amer. Derm. Assn.*, 1900).—Middleton, *Amer. Jour. Med. Sciences*, October, 1887.—Crocker and Williams, *Brit. Jour. Derm.*, vol. vi, 1894.—Bury, *Illustrated Med. News*, May 18, 1889.—Hutchinson, "Illustrations of Clin. Sur.," vol. i, 1878.—*Brit. Jour. Derm.*, vol. i, 1888–89.—*Archives of Surgery*, vol. i, 1889–90, vol. i, 1890–91.—Crocker, "Diseases of the Skin," 1904, p. 142.—Stelwagon, "Diseases of the Skin," 1902, p. 158.

Etiology and Pathology.—In Hutchinson's cases especially, and in some of the others, gout or acute rheumatism has apparently borne a relationship to the condition. In Bury's case there was intermittent albuminuria. The age and sex have been variable. It has been seen in both sexes and at varying ages, from childhood to later life.

There is no evidence that the disease belongs in the erythema group, and it is described here because of its name and for lack of any knowledge of its proper place.

Crocker regards the lesions as connective-tissue hyperplasias of inflammatory origin, analogous to subcutaneous rheumatic nodules, and not true neoplasms. Examination of a lesion excised from his and Williams' case showed that the process was seated in the deep portion of the corium and consisted of the development of a fibrocellular structure which had in great part replaced the normal fibers of the corium.

Treatment.—The cases which have thus far been studied have apparently been uninfluenced by treatment. The pathology suggests that x-rays might remove the lesions.

IV.—PELLAGRA[1]

SYNONYMS: *Lombardian leprosy, Endemic erythema, Mal rosso.*

Pellagra is an endemic, trophic disease of toxic origin, occurring in persons who habitually eat diseased maize, and characterized by gastrointestinal, cerebrospinal, and cutaneous symptoms.

The disease comes on gradually, nearly always in the spring, with weakness, articular pains, giddiness, headache, and severe burning pains in the back which radiate to the extremities and are especially noticeable in the feet and hands. With these there are symptoms of gastrointestinal catarrh, furred tongue, tense and painful epigastrium, loose bowels, and sometimes jaundice. These manifestations of the disease may precede by weeks the appearance of the eruption.

Eruption.—The skin symptoms appear suddenly, often within twenty-four hours, as a diffuse erythema of either a dark or bright shade of red. This may be simply a hyperæmia which disappears on pressure, or a livid congestion with or without hæmorrhage into the skin. *Bullæ* occur in rare cases, and are followed by indolent erosions. The skin is swollen and burns and itches severely. After about two weeks the erythema subsides and desquamation of the epidermis occurs, first in large flakes, afterwards in branny scales. Upon the disappearance of the active process the skin is left pigmented and somewhat thickened. Repeated attacks of the disease occur from year to year, and with the subsidence of each attack the pigmentation and the thickening are increased, and this continues for several years. After four or five years the thickened skin begins to undergo atrophy and takes on the characteristics of the senile skin; keratoses like senile keratoses form, and the skin becomes thin, loose, dry, wrinkled, and pigmented. As the disease continues from year to year, it becomes more extensive, and the atrophic changes may ultimately extend all over the body. At the same time, with these objective changes there may be also some loss of sensibility in the affected areas.

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[1] Scheiber, "Ueber Pellagra in Rumänien," *Archiv*, 1875.—Houchard, "Expériences relatives à la production de l'érythème solaire et plus particulièrment de l'érythème pellagreux," *Comp. Rend. de la Soc. de Biologie*, 1877.—Hardy, "De la pellagre," *Gaz. des Hop.*, 1882; translation in *Jour. Cut. and Gen.-Urin. Dis.*, 1883, p. 123.—Neusser, "Die Pellagra in Oesterreich und Rumänien," Vienna, 1887.—Raymond, "Les altérations cutanèes de la pellagre," *Annales*, 1889, p. 627.

The distribution is one of the most characteristic features; the eruption is confined to parts which are exposed to the sun, the backs of the hands (the most characteristic location), the forearms, face, neck, and dorsa of the feet. It appears first on the backs of the hands. Beginning in the spring, the disease lasts until July or August and then declines. It may entirely disappear in the winter to reappear the following spring, usually with aggravated intensity. After two or three years all of the constitutional symptoms become exaggerated. The tongue becomes red and bald, there is a burning sensation in the mouth, swallowing is painful, diarrhœa increases, and the patient emaciates rapidly. There are severe headache and backache, tenderness over the dorsal vertebræ, and insomnia. Paralysis of the third nerve is common, as are changes in the fundus oculi. The reflexes are at first increased and later diminish or disappear. In the late stages of the disease all of the cerebrospinal symptoms are increased. During the course of the disease mental disturbances are common: melancholia, insanity, or imbecility. Occasionally there are epileptiform convulsions, paralyses of various muscles, and other manifestations of degenerative nervous processes. In mild cases the disease may last ten or fifteen years. The average duration is about five years.

Etiology and Pathology.—The two apparently essential factors in the production of the disease are the habitual use of diseased Indian corn and exposure to sunlight. The causative factors in the production of the disease are probably toxins which are developed by mold fungi in the decomposition of damp Indian corn, particularly that which is gathered before it has matured. Lombroso has produced symptoms resembling pellagra by the administrations of these toxins. The condition is thus analogous to ergotism. The rôle which sunlight plays in producing it is probably that of an exciting cause. As the result of altered trophic conditions the skin is changed, but it requires the irritation of the actinic rays of light to produce an actual pathological condition. The disease occurs endemically, and only in Italy among poverty-stricken peasants who in their labor in the fields are exposed to the sun and whose diet consists largely of polenta, a sort of porridge made from poor cornmeal.

Roussell has described what he calls a pseudo-pellagra which occurs in alcoholics with peripheral neuritis and is seen among the demented and general paralytics in asylums.

Pellagra occurs in children, but is most frequent between the ages of thirty and fifty. It is not contagious nor hereditary. It was first described in Spain in 1735, but northern Italy is its best known territory.

It is endemic to a large extent in parts of Austria, Spain, Portugal, Roumania, Corfu and Egypt, and it is not uncommon in all of the countries on the shores of the Mediterranean, in Europe, Asia and Africa. It occurs also in India, various parts of Africa, the Barbadoes and probably other West Indian Islands, Mexico and South America. Until recently pellagra was thought not to exist in the United States. Sherwell, in 1888, reported cases which he had recognized in Brooklyn in Italian sailors. Sherwell again, in 1902, called attention to a case of pellagra in an Italian immigrant. The first American case was reported by Harris, of Georgia, in 1902. This case presented the classical symptoms of the disease, and Harris recognized it as possible pellagra. Serious attention, however, to its presence in this country was first attracted by Searcy who, in 1907, described an epidemic of eighty-eight cases of pellagra occurring in the Mount Vernon, Alabama, Hospital for colored insane. Similar cases had been observed in the same hospital since 1901. Since Searcy's report cases or epidemics have been described in many parts of the South and in other parts of the United States—in Texas, Arkansas, South Carolina, North Carolina, Virginia, Georgia and Illinois—and there is no doubt that it is extensive in certain parts of the United States. Most of the cases have been observed in institutions, particularly those for the insane, where it is probable most of them collect an account of their mental symptoms, after originating and developing elsewhere.

Post mortem there are found atrophic or degenerative changes in most of the tissues. The changes in the nervous system, according to Crocker, are inflammatory changes in the cerebral and spinal membranes, fatty degeneration of the vessels and cells of the spinal cord, and primary lateral sclerosis in the dorsal region only. In the beginning the skin

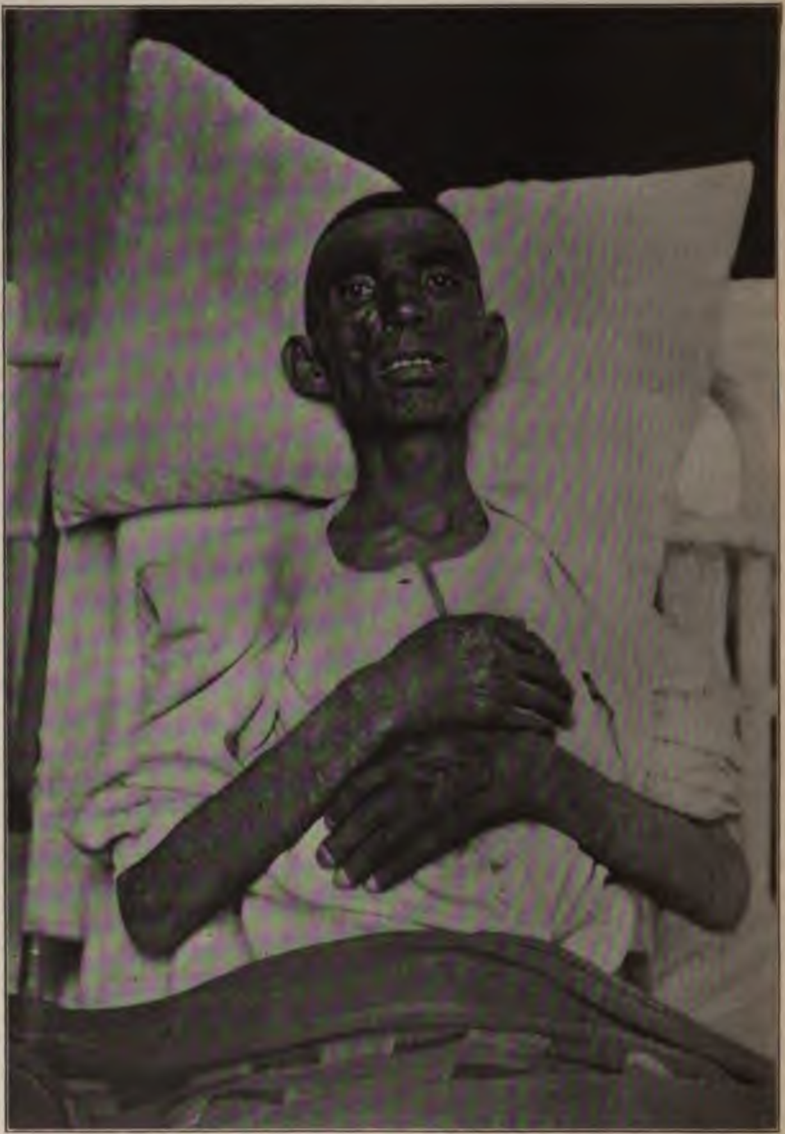

FIG. 48.—PELLAGRA. Bulgarian, age thirty-five, in this country about six months. Face pigmented. On back of hands sharply defined areas of dark, mahogany-brown color, horny epidermis thickened, roughened, and cracked. Mentally affected. Patient tuberculous (pulmonary and abdominal) and very cachetic. In service of Dr. Moore, Tuberculosis Hospital, Dunning, Chicago, July, 1909. (Author's collection.)

shows, according to Raymond, the changes of a mild inflammatory process; later there are the changes which are ordinarily found in the senile skin, atrophy of the rete, hyperkeratoses, and increased pigmentation.

Diagnosis.—The diagnostic feature of the disease is the triad of gastrointestinal, nervous, and cutaneous symptoms of distinctly toxic type. There is a chronic intestinal catarrh with red smooth tongue, salivation at times, indigestion, and usually diarrhœa. The absence of swelling or hemorrhage of the gums distinguishes it from scurvy. There are all degrees of mental disturbance or dullness, usually of melancholic type, up to insanity or dementia. The skin lesions are those of a toxic recurrent erythema, appearing upon the backs of the hands, the lower forearms, the face, and the dorsal surfaces of the feet. The recurrent attacks of erythema are followed by desquamation and result in pigmentation. There is more or less bronzing with roughening and cracking of the horny epidermis. The involved areas of the hands usually have sharply defined borders and stop abruptly at the sides of the fingers where the fingers come in contact. The skin symptoms are the most characteristic symptoms of the disease. They are unique and in the well-defined cases should not readily be confused with any other dermatosis.

Prognosis.—In patients in whom the disease is of slight intensity, and who have not had more than one previous attack, recovery may take place upon removal to favorable surroundings, but even then there is apt to be permanent damage to the nervous system. In advanced cases the prognosis is bad.

Treatment.—In beginning cases relief from the conditions which are recognized as predisposing to pellagra is the most important matter of treatment. In pronounced cases treatment is, as a rule, reduced to relief of symptoms and the support of the patients. The patients should be placed under favorable hygienic conditions and should particularly be given a suitable simple diet with avoidance of food prepared from questionable maize. A diet suitable to chronic enteritis is indicated. Medicinal treatment is for the most part symptomatic, with particular reference to the upbuilding of the patient. Iron, quinine, strychnin and, especially, arsenic are recommended by different authorities. Dyer regards quinine as of great value in the treatment. For the diarrhœa, opium is recommended. Purgatives are regarded as harmful and intestinal antiseptics of no value.

The local treatment of the cutaneous lesions consists in measures to keep clean the ulcerated or abraded surfaces and bland ointments to protect and soften the harsh and irritated skin.

V.—ACRODYNIA[1]

SYNONYM: *Erythema epidemicum.*

Acrodynia is a disease analogous to pellagra which was first observed in Paris in 1828 and 1830 by Chardon and Alibert. A few epidemics of it have been observed since, usually in soldiers and prisoners living in unhygienic surroundings and having bad food.

Like pellagra, the disease exhibits gastrointestinal, cutaneous, and nervous symptoms. Unlike pellagra, however, it runs a short course and is usually followed by recovery. The disease is ushered in by gastrointestinal disturbances, with nausea, vomiting, and diarrhœa, and these are more or less in evidence throughout the course of the disease.

When the issue is fatal it is chiefly caused by the weakness resulting from failure of nutrition and from diarrhœa. With the appearance of the gastrointestinal symptoms the hands and feet and face become swollen and the eyes suffused. This is quickly followed by patches of erythema upon which vesicles and bullæ develop. As the erythema subsides there is exfoliation of the epidermis, leaving the patches thickened and darkly pigmented. The eruption by preference involves the feet and hands, especially the palms and soles. It may spread over the limbs, and the chest and abdomen may also be involved. With the beginning of the disease there is noted formication and tingling and burning of the extremities with hyperæsthesia and severe pain in the hands and feet—a feature sufficiently characteristic to have suggested its name. This is followed by anæsthesia. In severe cases the limbs may waste and become œdematous, and there may be pareses and toxic spasms. The disease is afebrile, and runs a course of a few weeks to a few months. Recovery usually takes place except in the old and feeble.

The etiology and pathology of the disease are uncertain. It is apparently a toxic disease, arising from poisonous substances developed in decomposed grains of different sorts. Marquez has called attention to its similarity to chronic arsenical poisoning.

Its treatment is symptomatic.

VI.—PURPURA

Purpura in its essential characteristics belongs in the erythema group. It is a toxic condition with lesions similar in the mechanism of their production to the lesions of erythema multiforme, but with hæmorrhage into the lesions as a characteristic, instead of an exceptional, feature of the process. It, accordingly, should be studied with an appreciation of its relationship to erythema multiforme, although its consideration is deferred now, in order that all of the hæmorrhages of the skin may be considered together.

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[1] Alibert, "Monographe des Dermatoses," 2d ed., 1833, p. 12.—Tholozan, *Gaz. med. de Paris*, 1861, p. 647.—Marquez, *Gaz. hebdom.*, 1889, p. 91.

VII.—URTICARIA[1]

SYNONYMS: *Nettle Rash, Hives.*

Urticaria is an affection of the skin of which the salient characteristic is the occurrence of peculiar lesions called wheals. The description of wheals therefore covers in large part the objective symptoms of urticaria.

A wheal is a circumscribed elevation of the skin due to œdema of the corium. A typical wheal is the lesion produced in the average individual by the bite of a mosquito or a flea. It is slightly elevated, sharply circumscribed, with a flat or rounded surface, and of irregular outline. It may be of a pinkish-red color; when well developed, however, the center is usually pale and bloodless, but around this pale center there is an erythematous halo. A typical wheal is distinctly œdematous and has a soft œdematous feel, and the pressure of the edge of the finger nail causes a slowly disappearing pitting, as in other œdema. The development of a wheal takes place suddenly. There is first an erythematous spot which itches, and upon this the wheal quickly develops. The whole evolution of the lesion may occur in a few seconds or minutes. The duration of an individual lesion is usually short, from a few minutes to an hour or so, although occasionally lesions last for one or two days, or in certain cases much longer. Upon the subsidence of a wheal there is no desquamation, and, if there has been no traumatism from scratching, no trace is usually left. There may be slight pigmentation, and if, as frequently happens, there has been vigorous scratching, inflammatory papules or excoriations remain as the result of the traumatism. It is interesting to note that the scratch marks are indicative of the shape of the preceding wheal. If the wheal has been small and round, like an ordinary inflammatory papule, in scratching it the tip only will be torn off, leaving a punctate excoriation. If, however, as is usually the case, the lesion is large and flat, the scratch will extend across the entire lesion, leaving a linear excoriation which corresponds to the width of the wheal.

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[1] Török and Hari, *Archiv*, B. 65, H. 1, 1903.—Paramore, *Brit. Jour. Derm.*, 1906, vol. xviii, Nos. 7 and 8.

The articles on urticaria as well as those on pityriasis rubra, pityriasis rosea, and tattoo marks are adapted from articles contributed by me to "Reference Handbook of the Medical Sciences." For permission to reproduce the articles I am indebted to Wm. Wood & Company.

As would be expected of so capricious a lesion as a wheal, it appears with numerous variations, and accordingly several subvarieties of urticaria are described; the differences among these varieties, however, are for the most part unessential and their names are useful chiefly for convenience of description. In color wheals vary from pinkish to waxy white. If the œdema is intense enough to press out the blood from the capillaries, the wheals have a pale, waxy center with a narrow red halo at the border. Where the œdema is not so intense, or where the looseness of the tissue prevents extreme pressure from the œdema, the lesions are red as evidence of the hyperæmia. Wheals vary greatly in size. Typical wheals are usually from the size of half a finger nail to a twenty-five-cent piece. At times they are not larger than small papules, and may indeed be almost indistinguishable from inflammatory papules. There is practically no limit to their maximum size. They may be from the size of a silver dollar to a hand, and I have seen a wheal the elevation of which was not greater than a quarter of an inch which was certainly a foot wide and extended more than halfway around the trunk (*urticaria gigans*). Their shape is equally uncertain. Usually of more or less oval outline, they may be quite irregular and remind one of the outline on a map of an irregular island. When they occur in tissues like the prepuce and eyelids, which are lax and offer little resistance to the outpouring of serum, wheals often form tumor-like œdematous swellings that resemble a simple inflammatory process in the same tissue (*urticaria tuberosa*). They are differentiated by their sudden appearance and equally sudden subsidence, the presence, usually, of other ordinary urticarial wheals, and the absence of pain and other symptoms of acute inflammation. Very rarely the pressure of the extravasated fluid in the skin in urticaria is sufficient to cause uplifting of the epidermis so that the wheal is capped with a vesicle or bulla (*urticaria bullosa*). In still rarer instances the extravasation of serum is accompanied by enough red blood corpuscles to make the contents of the vesicle or bulla hæmorrhagic (*urticaria hæmorrhagica*).

It is not an infrequent experience to see the vasomotor mechanism so delicate that wheals may be produced at will by friction or slight traumatism (*urticaria factitia*). In cases of acute urticaria a certain degree of this phenomenon is usual and is a diagnostic symptom. It is elicited by running the finger nail or the end of some blunt object over the skin with a little force. The normal reaction produced by this is a red line. In urticaria, however, there is produced in addition to the red line a pinkish or pinkish-white œdematous ridge down its center. In certain individuals this vasomotor instability is inherent, so that factitious wheals may be produced at will. In such cases it is possible to produce wheals of any shape, so that figures or letters or more or less elevated designs may be written in wheals upon the skin (dermographism). In the extreme type of this vasomotor instability slight traumatism produces bullæ; this condition, while closely allied to congenital dermographism, is usually described as an independent condition, epidermolysis bullosa (*q. v.*).

An attack of urticaria may consist of only a few wheals or their number may be innumerable. They occur on any part of the body, without symmetry and without any regularity of distribution.

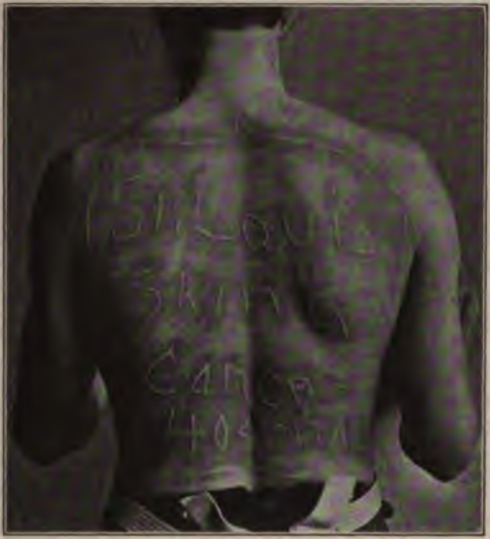
FIG. 49.—DERMOGRAPHISM.
(Engman and Mook's collection.)

A word further should be said concerning some of the subvarieties of urticaria spoken of above. The peculiarities of *urticaria tuberosa* depend almost solely upon its location at sites where the skin is lax, and the lesions deserve no special mention except to call attention to their occasional somewhat confusing appearance. The lesions of *urticaria bullosa* and of *urticaria hæmorrhagica* are usually seen in run-down individuals living in unfavorable surroundings. *Urticaria bullosa* occurs most frequently in children. *Urticaria hæmorrhagica* is frequently associated with other evidences of hæmorrhagic tendency, as hæmorrhages from the mucous orifices, and is an evidence of a profound systemic disturbance. Urticaria bullosa and, especially, urticaria hæmorrhagica are rare. The term *giant urticaria*, as indicated above, is applied to urticaria in which there is the development of wheals of very large size. These may be typical wheals, but frequently they are rapidly developing œdematous swellings which bear very slight, if any, resemblance to typical wheals. They occur most frequently about the face and extremities, and often form tense, angry-looking swellings that may suggest an acute cellulitis and that for the time cause alarming distortion. When occurring on the hands or feet such lesions may cause tense, diffuse œdematous swellings which will put the part on the stretch and lead one to suspect an acute inflammatory process. Frequent sites of these lesions are the eyelids, the cheeks, the nose, the lips, and the ears, and on such parts they may cause the most grotesque deformities. In all forms of urticaria evidences of lesions on the mucous membranes occur, and such lesions in giant urticaria are not infrequent. Giant urticaria may involve the tongue or the uvula, distending them enormously, or the epiglottis, producing alarming dyspnœa. This dyspnœa may cause great distress, but very rarely or never does it so interfere with breathing as to demand tracheotomy. The lesions of giant urticaria may occur singly, but several large lesions may occur at one time, and it is not infrequent for them to be associated with typical urticarial wheals. These lesions, like other urticarial wheals, develop rapidly, usually in the course of a few minutes, and after persisting for a short time, usually not more than a few hours, rapidly subside. They are painless, may or may not be accompanied by itching, and unless they mechanically interfere with some important function, are the source of little discomfort. There may be slight constitutional disturbances, such as occur in other forms of acute urticaria.

Giant urticaria is frequently described as a separate disease under the title of Angioneurotic Œdema, or Acute Circumscribed Œdema. It presents, however, no essential difference from other forms of urticaria. Its etiology, its course, and the fact of its being frequently accompanied by ordinary urticarial lesions, show its essential relationship to urticaria, and for that reason it is included here. As Osler puts it, it is urticaria "writ large." All that is to be said about etiology, pathology, prognosis, and treatment of other forms of urticaria applies with unessential variations to giant urticaria or angioneurotic œdema.

The original description of giant urticaria is usually credited to Quincke (*Monatshefte f. prakt. Derm. u. Syph.*, 1882). The condition, however, had been accurately described and its character recognized previously by Bannister (*Chicago Medical Review*, June 20, 1880).

The lesions of urticaria are characterized by more or less itching. If they are in lax tissues where swelling does not cause much tension the itching may be slight or entirely absent; ordinarily it is severe and, where the lesions occur upon parts like the hands, which are highly sensitive and in which from their structure the swelling is very tense, the itching is almost intolerable. The itching usually precedes for a few minutes the appearance of the wheals, persists during their continuance, and is somewhat paroxysmal in character. It may occur only at the site of the wheals, but if it is at all severe it is excited reflexly at other points. Not infrequently one sees the itching of urticaria occur independently of the eruption of wheals, *urticaria subcutanea*. This may occur in a predisposed individual independent of an outbreak, or it may precede an outbreak or follow it by several days.

Urticaria is a manifestation of many systemic disturbances, and will present the symptoms of the conditions which produce it. Frequently there are no constitutional symptoms except such as follow overeating or accompany a slight gastrointestinal disturbance. It is usual for the patient with urticaria to have some feeling of malaise, and occasionally urticaria is accompanied by an acute febrile disturbance with rise of temperature to 102° or 103° F., nausea and vomiting, and more or less prostration, *urticaria acuta or urticaria febrilis*. At times it is accompanied by a gastric crisis which apparently indicates the involvement of the stomach in the same process.

An attack of urticaria usually extends over a few hours or at most a day or two, the disease passing away after the appearance of several crops of evanescent lesions. At times, however, the disease persists for weeks and months in successive outbreaks of the eruption, and in such cases is characterized as *urticaria chronica*, the term referring to the persistence of the process as a whole rather than to any peculiarity of the individual attacks. On the other hand, urticaria is persistent not only from the occurrence of repeated outbreaks of the eruption, but also at times because the lesions lose their evanescent character and persist for days or weeks (*urticaria perstans*).

The *urticaria papulosa* of children, described by English dermatologists, is the best type of persistent urticaria. The characteristics of this form are the persistence of the lesions and the fact that the true urticarial wheals are followed by inflammatory papules which may persist for several weeks. It is readily seen that the occurrence of several crops of indolent wheals, followed by inflammatory papules which persist for weeks, will produce an exaggerated picture of the urticarial skin. In such cases, as a result of scratching, we have inflamed papules, infected excoriations, and more or less dermatitis, resulting in thickening and induration of the skin. These cases in their most extreme type are strikingly similar to Hebra's prurigo. It is, indeed, not unlikely that they pass at times into prurigo.

Etiology.—Urticaria is one of the commonest of eruptions. It is, in its ordinary form, so trivial an affection and so generally known that medical advice is not often sought. It occurs at any time of life, but is most frequent in childhood. Among young children acute attacks of urticaria are very common, as a result of imprudent eating or of slight disturbances of digestion.

In most urticarias two factors stand out prominently in the etiology. The first is individual predisposition; the second is the presence of some toxic substance which disturbs the local vasomotor tone. There are other forms of urticaria besides those due to the disturbance of the vasomotor mechanism of the skin by toxic substances, but they are relatively few.

Underlying the development of urticaria from all sorts of causes is individual predisposition. In certain individuals urticaria can be produced by the most trivial causes, such as the crawling of a caterpillar upon the skin, or the ingestion of the minutest quantity of some particular substance. In other individuals its occurrence is unknown. So violent an irritant even as the sting of a bee may not produce the smallest wheal.

The causes of urticaria may be divided into local and constitutional. Among the local causes of urticaria are especially to be mentioned the bites of parasites, such as fleas, mosquitoes, the various forms of pediculi, bedbugs, and the stings of wasps and bees. The lesions may also be caused by contact with stinging nettle, jellyfish, or caterpillars.

The constitutional causes of urticaria are numerous, but they are largely comprehended under various forms of intoxication. In certain individuals the ingestion of a particular article of food or of a particular medicine is followed by the development of an acute outbreak of urticaria. Among foodstuffs that may cause urticaria are: oysters, crabs, lobsters, and other shellfish; pork, sausage and other similar highly seasoned meats; mushrooms, strawberries, tomatoes, cucumbers, and nuts. Among medicines: opium, chloral, the salicylates, copaiba, cubebs, quinine, turpentine, valerian, and various coal-tar products. The most frequently offending substances among the above are perhaps shellfish and strawberries, opium, quinine, copaiba, and cubebs. Another form of intoxication from without, which is now a very common cause of urticaria and is a connecting link between the causes of urticaria introduced from without and those developed within, is that from the antitoxins. Urticaria is the most frequent cutaneous manifestation following the use of diphtheria antitoxin. Somewhat similar are the urticarias which have been described from the absorption of hydatid fluid.

The forms of intoxication from within which may cause urticaria are numerous, but far and away the most important is intoxication arising from the gastrointestinal tract. Overeating, constipation, and defective elimination are the group of factors most frequently seen in the causation of urticaria. Disturbances of the liver, particularly those which are accompanied by the presence of bile in the circulation, are at times responsible for outbreaks of urticaria. And the conditions which are vaguely described under lithæmia, gout, and rheumatism, of which the essential characteristic is a disturbance of the balance between assimilation and excretion or perhaps the presence of suboxidation products in the blood, are frequently responsible for chronic urticarias. Urticaria is also at times a result of malaria, and Matas has reported a case in which outbreaks of urticaria seemed to take the place of the usual malarial attacks. Anæmia is a factor which frequently has to be combated in the management of urticaria, and it occurs also at times in connection with albuminuria and glycosuria.

After intoxication, in the etiology of urticaria, come the various factors which may be included under the head of the nervous causes. The condition is seen with considerable frequency as a result of the nervous disturbances accompanying disorders of the genito-urinary tract, in pregnancy, in menstruation, and during lactation. It is excited at times by violent emotional disturbances, as grief, the dread of some impending crisis, or the shock of surgical operations. In particularly unstable individuals it may occur from such slight mental disturbances as anger, confusion, or surprise. Its occurrence as the result of rectal worms, phimosis, and similar forms of local irritation seems to indicate that at times it is due to reflex nervous irritation. The occurrence of urticaria in connection with nervous diseases is well recognized. This is rather frequent in functional nervous disturbances like hysteria. It is rarer in connection with organic diseases of the central nervous system.

Pathology.—Urticaria is the most characteristic angioneurotic eruption. There is a temporary disturbance of the vasomotor control with dilatation of the capillaries of the part, local hyperæmia, and a rapid outpouring of serum into the tissues. The intense œdema thus produced shows as a swelling of the skin, which at first is pink from hyperæmia, but later shows as a pale, bloodless center surrounded by a halo of hyperæmia, as the pressure becomes greater and drives the blood out of the capillaries. Microscopically the tissues show the changes of inflammation with the factor of œdema more than ordinarily prominent.

In examination of the blood in urticaria Leredde has found polynuclear leucocytosis while the attack was active, and eosinophilia as the attack subsided. Lazarus has also found eosinophilia. Wright found deficient coagulability of the blood in urticarial subjects, and has suggested the use of calcium chloride, 20 grains or more, t.i.d., as in hæmophilia, to correct this.

Diagnosis.—The diagnosis of typical urticaria offers no difficulties, and, indeed, is usually made without the physician's aid. The rarer varieties of urticaria—urticaria hæmorrhagica, urticaria bullosa, and angioneurotic œdema—may lead to some confusion. In the first two types there are usually evidences of typical urticaria sufficiently manifest to give the key to the diagnosis. In angioneurotic œdema, or giant urticaria, the suddenness of its onset, its rapid development, the freedom from pain, and the accompanying evidences of typical urticaria usually distinguish it from cellulitis, with which it is most likely to be confused. The diagnosis of urticaria in itself, however, is the smallest part of the problem with which one has to deal in these cases. The far more difficult part is the determination of the causative factors. This, in cases of chronic urticaria, often taxes one to the utmost.

Prognosis.—In acute urticaria relief is easy. In chronic urticarias the success of treatment depends altogether upon the difficulty which attends the relief of the underlying condition, and the prognosis as to ready relief in such cases should be very guarded.

Treatment.—The curative treatment of urticaria is altogether a question of the elimination of the underlying trouble, and the management of the various cases is along the line of rational therapeutics. In all cases the possibility that the disease may be due to local parasites should be borne in mind. Where any of these are found to be the cause, the treatment is of course that necessary for the destruction of the particular parasite.

The constitutional treatment of acute attacks of urticaria is usually simply a matter of the relief of the digestive disturbances which cause it. If the cases are seen early an emetic may be indicated. More frequently, however, a brisk cathartic, such as magnesium sulphate, is all that is necessary. The after treatment of these cases consists in keeping the bowels open, and placing the patients upon a careful diet until the digestive disturbance is relieved. Frequently an acute urticaria is the culmination of more or less continued digestive disturbance. In such cases the mercurial cathartics are particularly valuable.

In the chronic forms of urticaria permanent relief is usually the result of painstaking investigation into the causation. The questions of defective elimination, constipation, lithæmia, or some lack of balance between assimilation and excretion, particularly have to be taken into consideration. The first attention has to be paid to the digestion. The diet should be regulated, indigestible foods generally prohibited, and a bland, simple dietary followed for a time. The free use of fruits, contrary to the advice sometimes given, is in my opinion usually indicated, and the patients should drink plenty of water. Careful attention should be paid to elimination, especially by the bowels, and for this purpose the rather liberal use of aperients for a time is advisable. Of the aperients I think that calomel and blue mass easily rank first; after them come senna, podophyllin, aloin, cascara, sodium phosphate, and the aperient waters. In the gouty and lithæmic, alkalies and colchicum are useful. In stimulating elimination I have had excellent results from the daily hot bath, the patient staying in the bath for from ten to thirty minutes each day, so that free sweating occurs. In certain cases the nervous sedatives prove distinctly valuable; the bromides, acetanilid, phenacetin, and sulphonal are all at times useful.

It cannot be emphasized too strongly that in all persistent cases of urticaria the question of elimination and the condition of the digestive tract deserve first consideration. After that the other factors, such as malaria, jaundice, anæmia, and other less frequent causes, are to be investigated, and when found present are to be treated along the usual lines. Rational treatment failing, certain remedies used more or less empirically have been found helpful. Quinine, the salicylates, belladonna, and strychnine are useful in certain cases. Calcium chloride has been recommended by Wright, and at times it has seemed to me to be of service. Among the remedies used empirically, the one which has given me the greatest satisfaction has been pilocarpine. Given to the point of producing the slightest evidence of its physiological effects, it is valuable in relieving itching and giving the patients rest. In certain intractable cases without gastrointestinal trouble arsenic is recommended.

The local treatment of urticaria is almost entirely directed to the relief of itching. For this purpose in acute attacks, one of the commonest and most useful measures is the alkaline or acid bath. A general bath or sponge bath with sodium bicarbonate usually stops the itching immediately. Equally good results are frequently obtained with similar baths of common salt. Either the salt or the soda bath is most effective when it is rather strong. In the place of soda or salt, borax or ammonium chloride (one or two ounces to the bath) may be used with equally good effect.

Similar baths with vinegar or other acids are often equally effective, but they are not quite so agreeable, and usually nothing is gained by substituting them for the salt or soda bath. In place of the bath, alkaline or acid lotions may be substituted. As acid lotions, water and vinegar (equal parts), water two parts and vinegar one part, or alcohol two parts and vinegar one part, may be used with good effect. A one- or two-per-cent solution of carbolic acid with glycerine in water or in a boric-acid solution is a most excellent antipruritic in these cases. In place of the water the lotion is sometimes made more agreeable, and perhaps more effective, by the substitution of distilled extract of hamamelis. Other antipruritic lotions are made with compound tincture of coal tar, or camphor chloral, or resorcin in the strength of one to three per cent in water. A lotion which I have found particularly useful in severe itching, where the skin is not broken, is camphor chloral and carbolic acid, each one to three per cent, in equal parts of distilled extract of witch-hazel and alcohol. This may be made more cooling and more powerful by the addition of one to two per cent of menthol. In certain cases the addition to these lotions of an insoluble powder in suspension is advantageous. For this purpose suitable powders are zinc oxide and calamine. These may be added to such lotions as have been suggested above in the strength of one or two ounces to the pint. Calamine lotion is also an agreeable application. Frequently a most agreeable application, in conjunction with these other measures, is a dusting powder to be applied after the bath or after the use of a lotion. Suitable dusting powders for this purpose are zinc oxide, starch, talcum, or stearate of zinc. A useful addition to these powders is boric acid in the proportion of one half to one dram to the ounce, or salicylic acid, resorcin, or thymol in the proportion of five to twenty grains to the ounce. Usually, unless there is a secondary dermatitis, ointments are not desirable. They are mussy, inconvenient, and dirty, and not more effective than the lotions. Where, however, there is a secondary dermatitis as a result of scratching, ointments are frequently useful not only as vehicles for the active medicament, but as a protection for the abraded surfaces. In such conditions the antipruritic lotions have to be used with some care, as they are more or less irritating. Baths are not contraindicated, but the addition of bran or oatmeal to the baths in sufficient quantities to make them slightly mucilaginous renders them more soothing.

VIII.—URTICARIA PIGMENTOSA[1]

SYNONYM: *Xanthelasmoidea.*

Urticaria pigmentosa is a rare affection characterized by the development of wheals which are followed by peculiar persistent pigmented macules, papules, or nodules.

The essential process of the disease is the development of wheals which subside and recur upon the same spots with the formation of pigmented lesions as a result of the repeated recurrence. Two forms of the disease exist—the macular and the nodular. These may occur separately, or more frequently the disease presents a combination of the two types. The first evidence of the affection in some cases is the appearance of erythematous spots, which are followed in a day or two by the development of small reddish wheals. In other cases wheals develop suddenly without any noticeable premonitory erythema. In the macular form, upon the disappearance of the wheals, there are left fawn-colored or brownish-yellow pigmented stains. These are level with the skin or perhaps slightly elevated. In the nodular form there is the sudden eruption of wheals which are succeeded by nodular lesions that remain indefinitely. These are at first reddish, but in time become brownish-red or brownish-yellow. Similar lesions continue to appear, and so the disease goes on for many years. On these lesions bullæ may develop. Individual nodules may persist for years; at other times they shrink and disappear, leaving brownish, pigmented, and at times atrophic spots. Usually after several years new lesions no longer develop, and the disease gradually disappears. In both the macular and the nodular forms factitious urticaria can be produced by friction, and in the same way wheals may be made to develop at the site of the nodular or macular lesions. This formation of wheals upon the pigmented lesions by friction is a peculiar and characteristic feature. Itching in the cases is usually severe, and, continuing as it does for years, is the most serious feature of the disease.

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[1] Raymond, *These de Paris*, 1888 (complete review).—Elliot, *Jour. Cutan. Dis* 1891, p. 296.—Morrow, *ibid.*, 1895, p. 445.—Gilchrist, *Johns Hopkins Hosp. Bull.*, vol vii, 1896, p. 140.—Colcott Fox, *Brit. Jour. Derm.*, 1898, p. 411.—Brongersma, *Brit. Jour. Derm.*, 1899, p. 179 (Pathology).—Graham Little, *Brit. Jour. Derm.*, October November, December, 1905, and January, 1906.

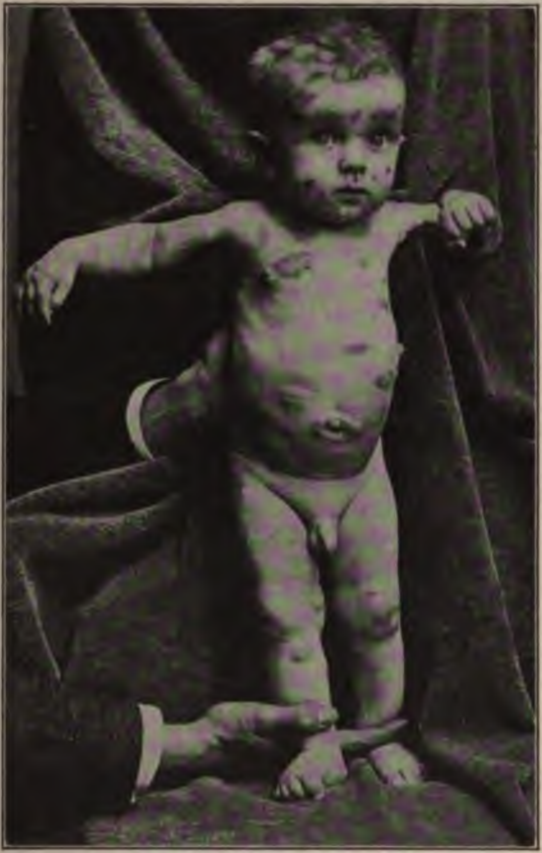
FIG. 50.—URTICARIA PIGMENTOSA, NODULAR TYPE. (Elliot's photograph.)

Ordinarily the eruption is abundant over the affected parts. In cases with large lesions the number may not be very large. Its distribution is general. It is usually most abundant upon the trunk and neck; then upon the limbs and head, and it is not apt to involve the palms and soles. Lesions have been seen on the mucous membranes of the mouth.

Etiology and Pathology.—The disease is extremely rare. It was first described by Nettleship in England in 1869, and it is apparently commonest in Great Britain, where chronic urticaria in children is also commonest. Of its actual cause we know nothing. The disease begins usually in the first few months of life. Nearly all the cases have begun under six months, and it may be congenital. In one of Crocker's cases the lesions were noticed at birth. A very few cases have begun later, two or three of them in adult life. The majority of cases has been in boys. In some of the cases severe gastrointestinal catarrh has existed.

Microscopically the lesions show no alterations in the epithelial layer except marked increase of pigment in the basal cells and some horizontal stretching of the epithelial cells. The upper part of the corium is œdematous, and there is a peculiarly abundant infiltration with mast cells. Unna was the first to call attention to the marked accumulation of mast cells in the corium, and his observations have been repeatedly confirmed by other observers. This abundant accumulation of mast cells is regarded as pathognomonic of the condition.

Diagnosis.—The characteristic features of the disease are the presence of persistent pigmented macules, papules, or flattened nodules which can be changed into wheals by rubbing. The itching and the fact that the wheals can be produced artificially suggest to one's mind the possibility of urticaria. The presence of these features and of the peculiar pigmented lesions should render the diagnosis easy. From ordinary chronic urticaria it can be differentiated by the peculiar persistent character of the lesions. Several cases have been published as persistent generalized xanthoma which, as later developments proved, were cases of urticaria pigmentosa. A differential diagnosis from xanthoma can be made by the fact that in xanthoma factitious wheals cannot be produced, and itching and other urticarial symptoms are absent. Xanthoma lesions also are of a distinctly yellow color; the lesions of urticaria pigmentosa may approximate this sulphur yellow, but are usually chamois color, dirty yellow, or brown.

Prognosis.—The disease usually persists for years, at least until the time of puberty, although some cases have been known to get well after a duration of one or two years.

Treatment.—Treatment has had no effect upon the course of the disease. Relief of digestive disturbances renders the disease less severe. The itching can in great part be controlled by the use of antipruritic applications such as are used in ordinary urticaria, and the local treatment is along the same lines as in urticaria.

Under the title, "Erytheme Miliaire Leucogenique Prurigeneaux Chronique," Miliam (*Annales*, 1906, vol. vii, p. 48; Abst., *Jour. Cutan. Dis.*, 1907, p. 128) has reported a case which is apparently an atypical urticaria pigmentosa in a man forty-eight years old.

IX.—PRURIGO[1]

SYNONYMS: *Strophulus prurigineux* (Hardy); *Scrofulide boutonneuse bénigne* (Bazin); *Juckblättern.*

Introductory.—Prurigo is a disease characterized by a persistent abundant eruption of recurrent, pale, inflammatory papules which itch intensely and result accordingly in the production of an exaggerated condition of scratched skin.

The term prurigo has been used more or less loosely as synonymous with pruritus. Good usage now, however, confines it to the symptom complex which was first described by Hebra.

The picture of prurigo consists in large part of secondary lesions which arise as a result of scratching in an intolerably itching dermatosis. Given a skin which, from whatever cause, itches on the extensor surfaces habitually year after year and so severely that violent scratching is excited, and the secondary lesions that would result would reproduce almost completely Hebra's prurigo. There has been much discussion, therefore, as to whether his prurigo is an individual disease. If it is an individual disease it must be sharply differentiated from closely analogous persistent secondary eruptions which result from habitual scratching, and in the following description of its symptomatology I have therefore followed closely Hebra's disciple and successor, Kaposi.

Symptomatology.—Prurigo begins between the eighth and twelfth months of life as an urticaria (Kaposi). This urticaria continues during the second year with the characteristics of the papular urticaria of children, the *lichen urticatus* of English authors; that is, there is much itching and insomnia, and there develop repeated and persistent recurrences of small papular urticarial wheals which, as a result of scratching, leave inflammatory papules with more or less dermatitis and secondary induration of the skin. According to Kaposi, the urticarial type of lesions continue only "into the second year," and in the beginning of the second year of life the characteristic pale, inflammatory papules make their appearance. But the description which he gives of this early stage of the papular eruption with the resulting lesions from scratching makes it impossible, in my opinion, to differentiate the condition from the papular urticaria so common in London, excepting only its somewhat characteristic distribution.

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[1] Hebra, vol. ii, p. 253.—Kaposi, p. 363.—Bernhardt, *Archiv f. Derm. u. Syph.*, 1901, vol. lvii, Nos. 1 and 2 (pathogenesis).—Robin, *Jour. d. pratic.*, 1902, No. 20 (Treatment).—Azuma, *Japinische Zeits. f. Derm. u. Urologie*, vol. ii, No. 2, April, 1904.—Corlett, "Prurigo in the United States," *Jour. Amer. Med. Assoc.*, July 30, 1904.—C. Fox, *Brit. Jour. Derm.*, March, 1905, p. 102.—Elliot, *Trans. New York Derm. Soci.*, October 24, 1905.—Whitehouse, *Jour. Cutan. Dis.*, April, 1906, p. 177.

By the beginning of the third year the typical picture of prurigo is developed. On the extensor surface of the legs, where the disease is most marked, there is an eruption of pale or red hard papules which project very slightly above the surface, give a nutmeg-grater feel to the skin, and are best appreciated by touch. Like urticarial wheals, these papules become larger on scratching. Many of them are excoriated and are tipped by a brownish crust of dried blood and serum. There are all the evidences of a dermatitis from the traumatism of scratching. The skin is thickened and indurated, showing in addition to the excoriated papules linear scratch marks and numerous pustules. The furrows are exaggerated, and from its thickening the skin is tense and rigid so that it cannot be picked up. It is markedly pigmented, and on scratching shows fine delicate scales. The intensity of the process varies: it is likely to improve in summer when perspiration is free; it is improved by placing the patient under better hygienic surroundings and by treatment; so that a patient presents very different pictures of the disease under varying conditions. With the development of the inflammatory lesions upon the legs there occurs swelling of the inguinal

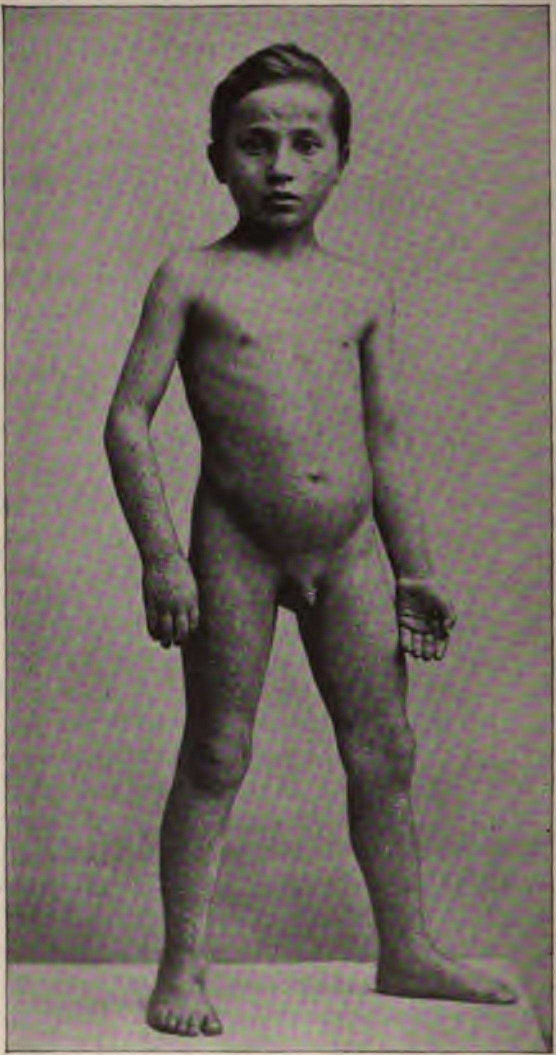

FIG. 51.—PRURIGO. (Zeisler, Morrow's System.)

glands, and this swelling, often very pronounced, remains a characteristic feature of the disease throughout its course.

The skin of these patients is always dry and rough, and they do not perspire on the extensor surfaces except when the condition is much improved. The hair is dry and lusterless, and on affected parts is broken off by scratching.

The foregoing is the type of severe prurigo, *prurigo agria seu p. ferox.* A milder type, *prurigo mitis,* characteristically milder throughout its entire course, was described by Hebra. The milder forms differ from the severer forms only by great mitigation of the symptoms in general. The papules are not so numerous, do not itch so severely, and, as a result, the sequelæ are less marked.

The distribution of prurigo is regarded as one of its most characteristic features. It occurs chiefly on the extensor surfaces of the limbs and increases in severity from above downward. It is most marked on the extensor aspect of the legs. On stroking the skin from the thigh downward there is distinct sensation of increasing roughness. After the legs it is most abundant over the sacrum and on the buttocks and extensor surfaces of the arms. Many discrete lesions occur over the trunk and a few upon the cheeks, neck, and forehead. A strong point is made of the fact that it does not attack the flexor surfaces of the large joints—the popliteal spaces, the bends of the elbows, the axillæ, and the groins. The dermatitis which occurs as a sequel of the process may extend onto the flexures, but the characteristic prurigo papules, according to Kaposi, are never found on the flexures. In prurigo mitis the distribution is much less extensive. It may be confined to the legs, exceptionally to the arms (Kaposi).

The disease ordinarily begins in early childhood and continues throughout life. It may be fully developed to the most extreme degree of prurigo agria within a few years after its appearance. Prurigo mitis when taken in early childhood may be cured, and prurigo ferox, even in later life, may be much improved by persistent treatment and improved surroundings.

Prurigo patients show the constitutional changes that result from an intolerable itching dermatosis which causes insomnia and severe nervous strain. They are poorly nourished and physically and mentally reduced.

Etiology and Pathology.—Prurigo, according to Kaposi, always begins between the eighth and the twelfth months of life. It is not present in the newborn. More recent observers agree that it may begin earlier than the eighth month and as late as the eighth or tenth year of age (Crocker), or even much later (from a few days to twenty-nine years of age, Ehlers). It occurs in males more frequently than in females. It is distinctly, like the chronic urticaria of children, a disease of the poor, with bad hygienic surroundings and bad food as the most important factors. There seems to be some hereditary predisposition, but the disease is not transmissible by inheritance. According to Hebra, it is often seen in children of tuberculous mothers. It is not due to external irritants and is not contagious. The disease is especially common in Hungary and Austria. It is more or less common in all European countries where the poor classes live in extreme squalor. It occurs in America very rarely, except in immigrants, and imported cases ordinarily improve upon residence in this country.

In my opinion, prurigo is closely analogous to the chronic urticaria of children, the chief difference being that, while lichen urticatus ordinarily gets well in the course of a few years, prurigo persists usually through life. Prurigo mitis, however, may get well in childhood, and the fact that prurigo may exist through life does not indicate any essential difference from papular urticaria, which may exist for a year or more. A more definite predisposition, the continuance of the exciting factors, and the impress made upon the nervous mechanism of the skin by a long-continued severe itching dermatosis, persisting even for a few years and establishing, as it were, a bad habit in the skin, would sufficiently explain the persistence of the disease. The primary eruption is an urticaria. And according to Unna and Riehl the primary changes in the prurigo papule are those of the urticarial wheal. All these considerations indicate its close relationship to urticaria. But whether it is a form of persistent urticaria or not, there is no question of the importance of the recognition of the clinical pictures.

According to Unna, Riehl, and Holden the anatomical findings indicate that the papule has an urticarial basis. There is dilatation of the blood vessels and lymphatic spaces with acute œdema of the corium, and perivascular round cell infiltration. There is proliferation of the rete cells, as in other inflammatory processes, and more or less increase of pigment in the rete and corium. The arrector pili muscles are contracted and hypertrophied (Holden), so that the hairs, if not torn out, are erected. Deep in the rete, vesicles are found which are not visible to the naked eye and which become pustules from invasion of leucocytes. In other words, the histological changes are chiefly those of an inflammatory process in the skin.

Diagnosis.—The history of beginning in early infancy and persisting indefinitely; the pale, hard, small inflammatory papules which characterize the disease; its distribution; the secondary inflammatory changes in the skin, with enlargement of the inguinal glands; the intolerable itching; the harshness and dryness of the skin, even where it is not involved in the process; and the sort of patient in which it occurs—these features make a characteristic syndrome. From papular eczema occurring with xeroderma it is distinguished by its history, the characteristic distribution, the presence of typical papules, and the extreme type of sequelæ. The history and distribution alone would suffice to make a diagnosis from ordinary urticaria and dermatitis due to parasites.

Treatment.—Treatment is directed to improving the general health, to the relief of itching, and to the removal of the eruption and its sequelæ. Much is gained by hygienic surroundings, liberal diet, and the use of tonics, especially of cod-liver oil and iron. According to Crocker, great improvement can be gotten by rest in bed and liberal feeding alone. Kaposi has seen benefit follow the internal administration of carbolic acid, and recommends pilocarpine for the temporary relief of symptoms. Cannabis indica in full doses is useful for the relief of itching (Crocker), as is antipyrine in small doses (Blaschko); thyroid extract is said to remove the eruption as long as it is taken (Dombrowski).

For the relief of secondary eczema and similar sequelæ the usual methods of treatment are indicated. After these are relieved great benefit is obtained from prolonged baths and the inunction of the skin with fats. A daily bath should be given of half an hour's duration at a temperature of about 90° F. Alkaline baths, such as sodium bicarbonate (two to four ounces to the bath), are best where the condition is acute. If the process is not too acute the bath is greatly improved by the addition of potassium sulphide (two to four ounces), or Vleminck's solution (two to four ounces). After the bath the skin should be anointed. This may be done either with vaseline or one of the fats alone, or with an ointment containing tar (five to ten per cent), sulphur (five to ten per cent), naphthol (two to five per cent). Kaposi especially recommends an ointment of naphthol (five per cent for adults, one to two per cent for children). This is rubbed in every night, and every other or every third night the patient is bathed with naphthol sulphur soap. This treatment usually produces rapid improvement.

X.—HERPES[1]

The term herpes (from ἕρπειν, to creep) was originally a generic term used in dermatology to indicate eruptions of various sorts of which spreading was a feature. Later it was taken by the French to denominate a supposed diathesis (Herpétisme) and the eruptions which characterized it (Herpétides). Such usages of the term are obsolete, and its adjectives, herpetic and herpetiform, are now used only to signify that lesions are like those of herpes simplex and herpes zoster—i. e., are grouped lesions, usually vesicles, on an erythematous base.

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[1] Merk, *Wien. klin. Wochenschr.*, 1903, No. 9.—Howard, *Amer. Jour. Med. Sci.*, February, 1903.—Kopytowski, *Archiv f. Derm. u. Syph.*, vol. lviii, pp. 55–80 and 387–482.

There are two substantive diseases which belong in the group of herpes: (1) Herpes simplex and (2) herpes zoster. These are both trophoneurotic dermatoses. The lesions of both are similar, but whether they are essentially related is a moot point.

HERPES SIMPLEX

SYNONYMS: *Herpes, Fever Blisters, Cold Sores.*

Herpes simplex is an affection characterized by the spontaneous development around the muco-cutaneous junctures of grouped vesicles upon an inflammatory base.

Herpes simplex occurs in two forms, *herpes facialis* and *herpes progenitalis.*

Herpes Facialis

SYNONYMS: *Herpes febrilis, Herpes labialis, Hydroa febrile, Cold Sores, Fever Blisters.*

Herpes facialis is familiar in the lesions known as cold sores or fever blisters. The first manifestation of the eruption is rather severe tingling, throbbing, burning, and a feeling of tension at the affected point, usually on the vermilion border of the lips. This is quickly followed by slight swelling and the development of a group of slightly reddened papules, which in the course of a few hours develop into a vesicle or a group of vesicles upon a reddened base. They may remain small, acuminate vesicles, but usually are rather large and rounded, and adjacent vesicles may become confluent, forming small, irregular blebs. The contents are first clear or translucent, then become turbid, and, if the vesicles are not ruptured, are gradually absorbed. The epidermal roof of the vesicle exfoliates, and a red stain is left which disappears in the course of a few days. The course of the lesions is rapid. They may develop fully overnight, but usually enlarge for twelve to twenty-four hours. After twenty-four to forty-eight hours absorption begins, and in the course of three to five days they dry up and exfoliate. The vesicles do not rupture of themselves, and if rupture does not occur they do not ordinarily become purulent. If through maceration or traumatism they are ruptured, the raw surface is likely to become infected, with the production of a small area of suppurating dermatitis. During the time of their development they are uncomfortable, and may be quite painful. The pain persists while they are tense, and disappears as absorption of their contents begins.

The commonest location of herpes facialis is about the mouth. Less frequently it appears around the muco-cutaneous juncture of the nostrils, and quite uncommonly upon the auricles. The lesions may also develop upon the skin at some distance from the muco-cutaneous junctures, so that practically any part of the face, except the forehead, may be involved. They occur most frequently as a single group of lesions, but in children the eruption is frequently extensive and may spread over a large part of the cheeks and lips and chin. Not infrequently they almost surround the mouth. When the lesions are numerous they show none of the tendency of zoster to be definitely confined to one side of the face.

They may also appear upon the mucous membranes of the mouth and conjunctivæ. In the mouth they are of not infrequent occurrence in connection with gastrointestinal disturbances. Here they appear as vesicles or groups of vesicles on a red base, which quickly rupture, leaving small ulcers that are painful out of all proportion to their extent. These lesions in the mouth are exactly analogous to the lesions about the genitals. It may be remarked here that herpes in the mouth is quite commonly described as a form of aphthæ, and is confused with true thrush or sprue, which is a specific disease produced by the *oïdium albicans.* The small painful ulcers resulting from herpes in the mouth may persist for several days if untreated. The pain disappears, however, and they heal readily if they are touched with nitrate of silver.

Etiology.—Herpes facialis is most frequent in children. After childhood I think it is more frequent in females than in males.

There is a marked predisposition to it in certain individuals, so that they may be annoyed by repeated recurrences up to adult life and even later. It can be excited in predisposed individuals by local irritation, such mechanical irritation as exposure to cold winds, sunlight, or sea air, and local traumatism. Hutchinson especially emphasizes its association with preceding chilliness. Herpes facialis is most frequently excited by some slight indisposition, a gastrointestinal disturbance, a slight cold, especially in children, or by acute febrile disturbances, more particularly bronchitis, the exanthemata, pneumonia, epidermic cerebrospinal meningitis, and malaria. It is occasionally associated with gout.

Apparently herpes is a trophic disturbance resulting from irritation in the terminal filaments of the nerves of the parts or of the adjacent sympathetic ganglion. The irritation may be of local origin, but it is most frequently due to toxic or other systemic disturbances. Savage and Seaton have described epidemics of herpetic fever characterized by a chill and more or less general disturbance and an abundant eruption of herpes.

Anatomically the condition is an acute inflammatory process beginning in the papillary layer and involving the rete with the formation of vesicles therein. Neuritis of the terminal nerve fibrils may occur.

Microörganisms have been isolated from herpes lesions, but the causal relation is uncertain, is indeed improbable.

Diagnosis.—The diagnosis of herpes facialis offers little difficulty. The sudden outbreak of a few tense, painful vesicles around the mouth or nose is characteristic. When the lesions become infected and pustules develop from inoculation, confusion with impetigo may take place, but a differential diagnosis is of no importance here because the condition is then really an impetigo which has had its origin in the opportunity for infection offered by the cold sore. Extensive simple herpes may be confused with herpes zoster. The points of difference are that herpes zoster is sharply unilateral; on the face it follows the distribution of the cutaneous branches of the fifth nerve, and is usually confined in distribution to one of these branches. In very rare cases, however, herpes zoster and herpes simplex on the face may so closely approximate the characteristic features of each other that a differential diagnosis is impossible.

Treatment.—The tension and burning can be relieved by puncturing the vesicles. The objection to this is that the lesions are then very apt to become infected.

Young women who are subject to herpes are usually very anxious to abort the lesions and use many things for this purpose. Spirits of camphor is, perhaps, most frequently used; it is a comforting application and is, I believe, of some service. Calamine lotion or solution of sulphate of zinc and potassium sulphide, each one to two per cent, are agreeable applications in the early stage, and some patients pin their faith to the latter lotion to abort the lesions. After the lesions are well developed they need no special treatment except measures to prevent their infection. If they are unruptured nothing is necessary; if ruptured they should be cleansed twice a day with solution of hydrogen peroxide or some other mild antiseptic, and be protected by a non-irritating ointment. The abraded surface may become fissured. Under these circumstances an excellent plan is to paint the lesions several times a day with tincture of benzoin or compound tincture of benzoin, either of which leaves a coating of benzoin over the surface. When the fissures are deep and are constantly kept open by the movements of the mouth, I have found it a very good device to apply a layer of collodion to the outer part of the fissure. This acts mechanically by preventing the tearing open of the fissure by motion, and healing quickly follows.

Herpes Progenitalis

SYNONYMS: *Herpes præputialis, Herpes genitalis.*

Herpes of the genitals is frequently described as a distinct affection from herpes of the face, because of the clinical differences which the two varieties manifest. In my opinion there is no essential difference between these two forms of herpes, the variations which herpes of the genitals presents being attributable to the fact that the lesions are always ruptured as a result of maceration, and present open abrasions which are likely to become infected. This form of herpes occurs on the glans penis and prepuce and on the vulva. It usually occurs upon the mucous surface, but it may occur upon the closely adjacent skin. The disease begins with the sudden appearance of a papule or a group of papules upon a tender erythematous base. The lesions, however, are not usually seen in the few hours before they have become vesicles. The vesicles quickly rupture as the result of maceration and leave a superficial abraded surface presenting a circinate outline corresponding to the base of the vesicles. If this is not infected it heals over in a few days, leaving a temporary stain. The abraded lesions are very apt to become infected, and are then converted into small suppurating ulcers and run the course of such a lesion.

With both herpes facialis and herpes progenitalis enlargement of the contiguous glands may occur.

Etiology.—This form of herpes is most common in male adults. Predisposition is a marked factor in its etiology. It occurs repeatedly in certain individuals, perhaps every few weeks or months, for years. It may be produced by such indispositions as are associated with herpes facialis, but is usually excited by local irritation. It is most frequently noticed after coitus, and may recur with menstruation. It is said in most cases to follow by a few weeks the disappearance of a venereal disease, but there is no reason for believing in such an association.

Diagnosis.—The characteristic lesion of genital herpes is an abrasion or group of abrasions representing the bases of a few vesicles which have coalesced into a small circinate figure. The ulceration, if any, is very superficial, unless there is secondary infection, and, if uninfected, the lesions run a rapid course, healing in five to eight days. The condition has to be diagnosticated from venereal ulcers. The hard chancre has a deep indurated base; it is usually a single ulcer of regular, not circinate, outline; there is some destruction of connective tissue by the ulcerative process, and the ulcer is indolent, lasting several weeks. The soft chancre is readily distinguished from the uninfected herpes. It is a deep inflammatory ulcer with probable infection of the adjacent glands. When herpes becomes infected with the formation of an ulceration and involvement of the adjoining glands it cannot, in many cases, be differentiated from the soft chancre. Indeed, it is no longer a herpes; the herpes was simply the starting point of the infection, and the resultant lesion is an infected ulcer analogous to a soft chancre and requiring the same sort of treatment.

Treatment.—The best treatment is the application of a small wet dressing of weak bichloride or boric-acid solution or some similar antiseptic. Nonirritating antiseptic powders, such as boric acid or aristol, are usually sufficient. If these are used it is well to prevent their caking by the occasional application of a mild antiseptic salve. As a prophylaxis galvanism is recommended, with the application of the positive pole over the lumbar region, the negative pole over the affected part, and a current of one half to one milliampère for ten minutes daily. Another recommendation is the application of a mustard plaster daily or every other day over the lumbar spine. Hutchinson advises the administration of arsenic to prevent the recurrence of herpes. The best means to prevent recurrence is the daily use of some astringent wash, such as zinc sulphate (one half to two per cent) or tannic acid (five to ten per cent in water). Cleanliness and the avoidance of irritation should be enjoined, and if there is a long prepuce it should be removed.

PERSISTENT BALANITIS

Crocker describes a recurrent superficial inflammation of the glans and prepuce which occurs in men between sixty and seventy, and in one case began as a recurrent herpes. It occurs as sharply defined superficial excoriations which persist or constantly recur and are likely ultimately to develop into epitheliomata. I have seen one case which corresponds to this condition which persisted for four years as superficial circumscribed areas of balanitis with excoriations. Ultimately typical epithelioma developed upon the sites, with rapid spreading to the inguinal glands and a very malignant course. The condition would seem to be analogous to other forms of precancerous lesions, like the first stage of Paget's disease or inflamed senile keratoses, and it is doubtful if it has any relation to herpes. Hutchinson regards it as incurable, but Crocker has been able to relieve it by the use of bactericides. He recommends bichloride solution, 1 : 6,000 or 1 : 8,000, applied as a wet dressing until irritation is produced, and then the use of boric-acid solution until the irritation subsides, and so on. He has also been successful by the application of a drop of pure formalin, an agent which would be effectual in a very superficial epithelioma. The effect of x-rays upon epithelioma and upon the usual precancerous lesions would suggest their use in the condition. A persistent or recurrent balanitis in one beyond middle life is always dangerous through the likelihood of its becoming epitheliomatous, and justifies radical measures for its relief.

HERPES ZOSTER[1]

SYNONYMS: *Zona* (a girdle), *Shingles* (from *cingulum*, a girdle).

Herpes zoster (from ζωστήρ, a girdle) is a disease characterized by the development of groups of herpetic vesicles in the area supplied by the nerve fibers from one or two posterior root ganglia.

Zoster is always associated with a lesion of a cutaneous nerve, and its salient characteristics are (1) grouped inflammatory papules or vesicles, whose distribution (2) corresponds with a cutaneous nerve area. It occurs most frequently in the course of the intercostal nerves, producing a girdlelike lesion, hence the unanimity of meaning in its various names. This, however, is by no means the only location of the eruption.

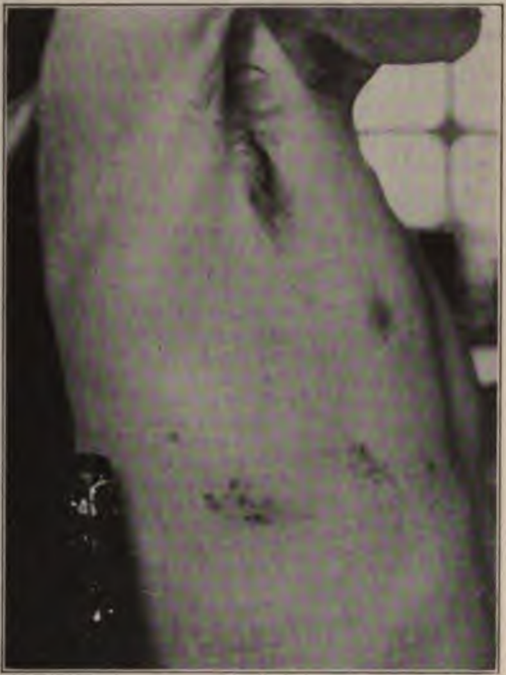

FIG. 52.—HERPES ZOSTER, SHOWING SIMPLY AS A FEW GROUPS OF PAPULO VESICLES IN THE COURSE OF AN INTERCOSTAL NERVE. (Author's collection.)

Symptomatology.—The eruption begins with the sudden appearance of a group of acuminate inflammatory papules. The eruption may not go further than the production of papules or of pinhead-sized vesicles on the tips of papules, but ordinarily after the appearance of the papules they quickly develop into tense vesicles. These are ordinarily large, up to the size of a pea or larger, and when, as usual, they are close together they coalesce into irregular bullæ. Their contents are at first clear or opalescent serum. This soon becomes turbid, but does not ordinarily become purulent unless there is secondary infection. In severe cases, however, they may become spontaneously purulent and occasionally even hæmorrhagic (*Zoster hæmorrhagica*). The vesicles do not usually rupture spontaneously, but after reaching their full development the contents are gradually absorbed and finally the roofs of the vesicles exfoliate, leaving red stains which may be pigmented and which persist frequently for some time. Ordinarily the process does not cause destruction of the corium, so that the lesions leave only stains in their track. Occasionally, however, the process is so acute that ulceration of the corium takes place, and this is followed by permanent scarring. Ulcerative zoster is particularly apt to occur in the region of the supra-orbital nerve. In the severest forms of zoster, gangrene may take place in the involved area and cause much destruction of tissue and ultimately disfiguring scars. Gangrenous zoster is a serious affection accompanied by severe pain. It is usually an evidence of greatly lowered vitality and may end fatally. It is not infrequently followed by pneumonia. It is not uncommon for the adjacent glands to be enlarged during the eruption of zoster; they may enlarge simultaneously with or before the eruption.

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[1] Kopytowski, *Archiv*, 1900, liv. 17 (Pathology).—Van Harlingen, *Amer. Jour. Med. Sci.*, January, 1902 (Etiology).—Winfield, *New York Med. Jour.*, August 2, 1902 (Malarial).—Eddowes, *Times and Hosp. Gaz.*, August 9, 1902 (Etiology).—Corlett, *Jour. Cutan. Dis.*, July, 1905, p. 289 (Varicella Accompanying).—Pollitzer, *Jour. Cutan. Dis.*, vol. xxi, February, 1903 (Histology).—Morrow, *Jour. Cutan. Dis.*, April, 1905, p. 157 (Ethyl Chloride in Treatment).—Evans, *Brit. Jour. Derm.*, 1905, p. 199, (age in).—Head and Campbell, *Brain*, 1900, xxiii, p. 333.

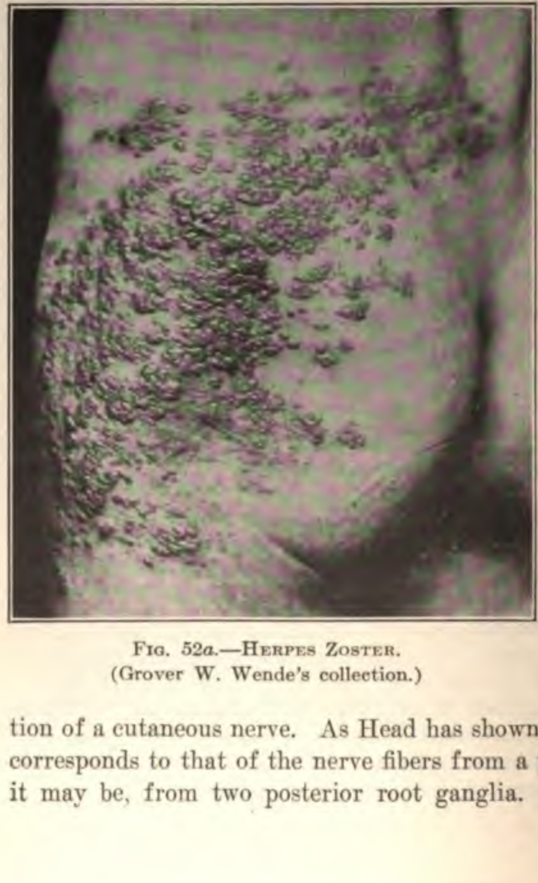

FIG. 52*a*.—HERPES ZOSTER.
(Grover W. Wende's collection.)

The duration of one crop of lesions from its appearance to the time of exfoliation is from a week to ten days, but by the appearance of successive groups of lesions the disease may be prolonged from ten days to three weeks. In the ulcerative and gangrenous cases the duration is much longer.

Zoster is usually said to follow the distribution of a cutaneous nerve. As Head has shown, however, the distribution corresponds to that of the nerve fibers from a posterior root ganglion or, it may be, from two posterior root ganglia. Apparently the fibers of more than one nerve pass through a single ganglion, and this accounts for the fact, long noticed, that the eruption may spread beyond the distribution of a single cutaneous nerve. The distribution is further complicated by the fact that at one time only a part of a ganglionic area may be involved, while at another it may spread over the areas of two adjacent ganglia. To speak accurately, therefore, the eruption corresponds to the distribution of the ganglionic areas, but bearing this in mind, it may be said for convenience that the distribution corresponds with that of the cutaneous nerves.

The entire eruption of a zoster may amount to only a small group of acuminate papules. Ordinarily it consists of two or more groups of large vesicles or small bullæ involving an area in the aggregate perhaps the size of a hand. At other times the entire area of distribution of the nerve fibers of a posterior ganglion or of two adjacent ganglia may be involved in an almost, but not quite, confluent eruption of large vesicles. Where there are several groups the eruption does not come out all at once, but in successive outbreaks, the first lesions appearing as a rule nearest the nerve center and the more distant groups later. The groups are likely to correspond in location with the points of exit of the cutaneous branches through the fascia. In an intercostal zoster one group is likely to be situated near the spine, another in the axillary line, and another near the median line in front (Crocker). Tenneson and others have called attention to the occurrence of "aberrant vesicles" on the same side with but at some distance from the original groups; these distant vesicles can frequently be found. When zoster spreads beyond the area of a single nerve ganglion, it involves contiguous areas. It is excessively rare for it to involve corresponding areas of the two sides, or for distant separate areas to be involved. It is therefore almost always asymmetrical. If the eruption is symmetrical it is said usually to be of syphilitic origin and to involve the trifacials.

Zoster may occur in the distribution of any cutaneous sensory nerve. The intercostal nerves are most frequently involved, and more than half the cases are on the trunk, and, according to Crocker, on the right side. It is not infrequent upon the face, where the Gasserian ganglion is involved. In this location it is most frequent in the area supplied by the supra-orbital nerve, and it is quite rare in the third division of the fifth. It is not common on the extremities, and is very rare below the elbows and knees. When the ophthalmic division of the fifth, especially the nasal branch, is involved, vesicles may develop upon the conjunctiva and cornea, and even iritis may occur. The corneal lesions are important. They are apt to be followed by scarring, and in severe cases there may be destructive ulceration of the cornea. When the second division of the fifth is involved lesions may develop upon the mucous membranes of the cheeks, palate, tongue, and tonsils. In rare instances disturbances of the adjacent motor nerves have been observed in zoster. This has been seen most frequently as zoster of the trifacial with paralysis of the third or seventh. Other combinations which have been seen are zoster of the second cervical with paralysis of the seventh cranial, and zoster of the first dorsal with paralysis of fingers and hand (radial and ulnar nerves).

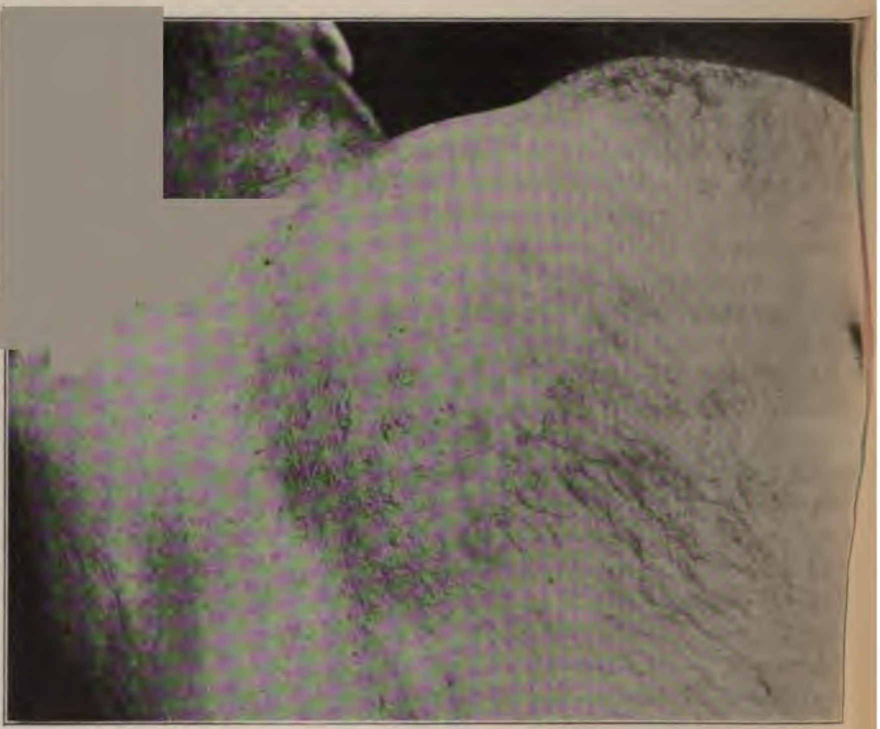

FIG. 53.—HERPES ZOSTER. Vesicles unusually small and closely aggregated, in patient with Pseudoleukæmia. (Author's collection.)

The characteristic subjective sensation of zoster is neuralgic pain. This pain varies in severity almost in direct proportion to the age of the patient. In patients under twenty it may be inconsiderable or almost absent. In old age it is often exceedingly severe. The pain usually precedes the eruption for a short time, from a few hours to several days, but in old people the neuritis and its pain may exist for several weeks before the eruption appears, and may continue for weeks or for months after its disappearance. The eruption itself is associated with tingling and burning and tenderness, which disappear with the beginning of involution of the lesions. Ordinarily there is no systemic disturbance, but occasionally there is fever.

Zoster very rarely occurs twice in an individual, but there are some exceptions. Grindon[1] has been able to find in the literature sixty-one cases of recurrent zoster. When zoster does recur it is usually the result of chronic peripheral irritation, as from a tumor or some other persistent pathological process involving the nerve.

Etiology and Pathology.—Zoster is rather a common disease, constituting one to one and a half per cent of skin diseases. It may occur at any age, but it is rare in infancy and most common in early adult life. Head's statistics show that seventy-five per cent of cases are under twenty-five. It is most frequent in spring and fall.

The essential factor in the production of zoster is irritation of a sensory nerve of the skin, and it may be caused by anything that will produce this. It sometimes results from mechanical irritation, such as a blow, the extraction of a tooth, a gunshot wound, the pressure of a tumor upon a nerve, or the irritation of a carious bone. An important exciting cause of zoster is exposure to cold draughts. Occasionally it is produced by severe mental emotions.

These reflex and mechanical causes of zoster, however, represent a small minority of the cases, and do not weaken the fact that in the large majority of cases the neuritis which underlies it is of toxic origin. As Hutchinson showed, it may result from the administration of arsenic, which at times causes peripheral neuritis. It is most frequently due to some infection. Indeed there is weighty authority for the opinion that it is a specific infectious disease. The grounds for this belief are its occurrence in epidemics, the fact that in epidemics it shows a more or less uniform type as regards severity, and the immunity which an attack furnishes. All the facts, however, seem to indicate that zoster is not an infectious disease due to a specific microörganism, but that it may be due to various infections. For example, the epidemic as well as most sporadic cases of zoster occur in the spring and fall, and are probably attributable to the various infections which excite the diseases of the respiratory mucous membranes that prevail in the same seasons. It is also seen at times to complicate such specific infectious diseases as epidemic cerebrospinal meningitis, as well as other forms of cerebral meningitis. And it is not infrequent in tuberculous subjects.

Zoster in nearly all cases is a manifestation of neuritis which usually involves the posterior ganglion of the affected nerve, but may attack the nerve beyond the ganglion and leave the latter unaffected. In rare cases it may be due to noninflammatory processes, such as hæmorrhage into a ganglion with subsequent degenerative changes.

The anatomical changes in the skin are those of an acute inflammatory process involving the corium and at times extending into the subcutaneous tissues. From the papillary layer it passes to the epidermis with the formation of, usually, unilocular, but sometimes multilocular, intercellular vesicles. Neuritis of the nerve filaments in the corium of the affected area has been demonstrated.

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[1] Grindon, *Jour. Cutan. Dis.*, 1895, pp. 191 and 252.

Diagnosis.—The occurrence of groups of large vesicles on an inflammatory base in the distribution of a cutaneous nerve, with neuralgic pains, forms a characteristic clinical picture which is not likely to be confused with anything else. When the eruption consists only of one or two groups of acuminate abortive vesicles the diagnosis may be obscured; here the key is furnished by the relatively severe character of the subjective symptoms and the distribution of the eruption. When occurring about the face it is most likely to be confused with simple herpes. This, however, is not as a rule so abundant, and if abundant is almost surely bilateral. Cases may arise with a unilateral eruption of herpetic vesicles in which a differential diagnosis is impossible. The diagnosis in such cases is not of practical importance. Indeed it is not absolutely certain that in the border-line cases there is any essential distinction.

Treatment.—Treatment is directed to the relief of pain and to the protection of the lesions. When the pain is severe its relief is the crying demand. In a debilitated patient tonics like iron, strychnine, arsenic, and cod-liver oil are useful; the patient should have a liberal diet, and in the winter should, if possible, be transferred to a mild climate. Otherwise he should be protected as far as possible against exposure to changes of temperature. When pain is moderate such anodynes as antipyrin, phenacetin, and aspirin give sufficient relief. In the severest cases hypodermics of morphine alone are efficient, and these are best given over the affected nerve. Other measures for relief of pain are blistering over the origin of the affected nerve and the application in the course of the nerve of a galvanic current of from two to five milliampères for ten minutes daily. More useful still for relief of pain is light freezing with ethyl chloride over the spine at the exit of the affected nerve. Much relief from the feeling of tension and the tenderness which accompany the early stage of the eruption can be obtained by puncturing the lesions, but if this is done care has to be exercised afterwards to protect the lesions from infections. Some comfort can be got from the application of cooling lotions, such as calamine lotion, or equal parts of alcohol and water, or alcohol with two per cent menthol. To any of these one half to one per cent carbolic acid is a good addition for its anodyne, as well as its antiseptic, action. These applications are to be dabbed upon the surface, which is afterwards covered with a protective dressing. If the eruption is on the trunk where a dressing can conveniently be worn, the best method of treatment is to dust it with boric acid, or some similar nonirritating antiseptic powder, and then cover the surface with a thick layer of absorbent cotton. Where a bandage is not convenient the necessary protection can be furnished by a fixed dressing. Unna's glycerine jelly is excellent for this purpose, and on small groups of lesions flexible collodion serves well. In lesions around the eyes where it is desirable to make every effort to prevent the spread of the eruption to the eye, patients should stay within doors and avoid all exposure. If this is impossible a thick protective dressing should be worn over the side of the face. When the lesions become ulcerated or gangrenous their local treatment is that of other ulcers.

XI.—DERMATITIS HERPETIFORMIS[1]

SYNONYMS: *Hydroa bulleux* (Bazin), *Hydroa herpetiforme* (Tilbury Fox), *Duhring's Disease*, *Dermatitis multiformis* (Piffard), *Herpes circinatus bullosus* (Wilson), *Pemphigus circinatus* (Rayer, Vienna School), *Herpes phlyctænodes* (Gilbert), *Pemphigus prurigineux* (Chausit, Hardy), *Pemphigus composé* (Devergie), *Herpes gestationis* (Milton, Bulkley), *Dermatite polymorphe douleureuse* (Brocq).

Dermatitis herpetiformis is a chronic relapsing disease characterized by a multiform eruption of inflammatory lesions which tend to appear in groups and are, as a rule, accompanied by an unusual amount of itching. It is a rare disease.

Dermatitis herpetiformis is a disease which presents many different clinical pictures, and various isolated cases of it have been described under different names before the recognition of their fundamental identity. By a brilliant generalization Duhring brought these various types of the disease together, and by a consistent description of its different phases established for it a place in dermatology. The recognition of the relationship of the different types of this very complex disease and their collection into one comprehensive syndrome is the greatest feat of American dermatology. The name dermatitis herpetiformis was suggested by Duhring as descriptive of the tendency of the eruption to occur in herpetic groups, and, in spite of various other names which have been suggested for the condition, Duhring's name remains the best and now has general acceptance.

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[1] Duhring, Cutaneous Medicine.—*Jour. A. M. A.*, August 30, 1884.—*Amer. Jour. Med. Sci.*, January, 1894, p. 93.—Various other papers all collected in the "Selected Monographs on Dermatology," published by the New Sydenham Society, 1893.—Brocq, *Annales*, 1888, vol. ix, and 1889, vol. x, series 3.—Jamieson and others, *Brit. Jour. Derm.*, vol. x, 1898.—Fordyce, *Jour. Cut. Dis.*, vol. xv, 1897.—Wende and Pease, *Jour. Cut. Dis.*, 1901.—Bowen, *Jour. Cutan. Dis.*, September, 1905 (Derm. herpetiformis in Children).—Bushnell and Williams, *Brit. Jour. Derm.*, May, 1906 (blood changes).—Engman and Mook, *Trans. Amer. Derm. Assoc.*, 1906.

All of the various forms of dermatitis herpetiformis have certain characteristics in common. The eruption is manifestly a vasomotor disturbance of internal origin. The eruption is polymorphic, and shows all types of inflammatory lesions from erythematous patches and wheals to bullæ and pustules, and even petechiæ may occur. None of the lesions, however, ulcerate except as the result of secondary infection. In different types of the disease one form of lesions may predominate, but more than one form is usually present in any outbreak. The lesions are widely distributed, and show a marked tendency to appear in groups and frequently to arrange themselves in ring formation and to spread peripherally. Like the lesions of herpes, the vesicles and bullæ of dermatitis herpetiformis do not readily rupture, and if uninjured resolve by absorption of their contents, the duration of a single lesion varying from one to two weeks. Upon the disappearance of a lesion sufficient pigmentation usually marks the site to make this feature a characteristic of the disease. The disease pursues a chronic relapsing course with exacerbations and remissions. During an attack the eruption does not pursue any definite self-limiting course, but groups of lesions develop at irregular intervals, so that an attack may continue for an indefinite time by the constant development of new crops of eruption. These attacks, however, are followed by intermissions or remissions and these by other exacerbations, and so on for months or years. An amount of subjective disturbance rather out of proportion to the intensity of the lesions characterizes the disease. This disturbance usually takes the form of intense itching. In some cases this is replaced by burning or a feeling of tension or positive pain.

Symptomatology.—The different forms of dermatitis herpetiformis are, according to the predominating type of lesions, erythematous, papular, vesicular, bullous, and pustular. For convenience of description o the various lesions these forms may be regarded as separate, but it mus be remembered that one of the striking characteristics of the disease i the multiformity of the eruption. One type of lesions may predominat and characterize the eruption, but in nearly all cases the eruption i mixed and lesions of different forms occur to a greater or less extent The most characteristic type of the disease is the vesicular.

Erythematous Type.—The erythematous eruption appears in ill defined or circumscribed patches of erythema from the size of a finge nail up to that of the hand or larger. The lesions are pinkish or reddish and are not perfectly flat but are slightly infiltrated, like the erythematous lesions of erythema multiforme, or like flat pinkish urticaria wheals, and they itch intensely. There may develop upon them maculopapules, vesicles, and bullæ. Where the erythematous patches ar sharply circumscribed they are usually of circinate outline. In thi the lesions begin as large flat maculo-papules, and spread peripher- with the formation of ringed lesions with a raised reddish border a flat erythematous center. These rings coalesce into circinate fig-

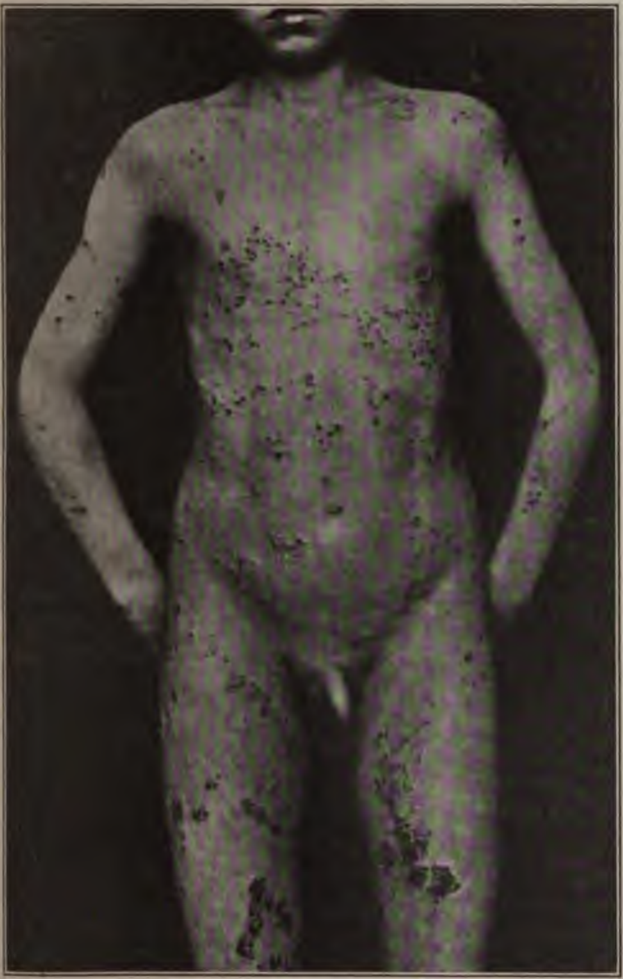

Fig. 54.—Dermatitis Herpetiformis. (Grover W. Wende's collection.)

s, thus forming figures which resemble the circinate form of erythema rginatum, but differ in that they itch intensely. The erythematous ons are frequently the first manifestation of the disease.

Papular Type.—The papular lesions occur as flat, ill-defined papular ltrations, resembling the abortive papular lesions of herpes zoster. y are of variable size, ordinarily larger than the papules of chronic ma, but, except for their arrangement in groups, presenting on the whole a similarity to chronic eczema. These lesions may remain as such and pursue a chronic course, lasting from one to two or four weeks, or vesicles may develop upon many of them.

Vesicular and Bullous Types.—In the vesicular form the lesions appear as clear, tense vesicles of varying size, from a pinhead to a pea, and at the start are without areolæ, but later they become turbid and areolæ develop around them. Their variable size is a striking characteristic; there may be vesicles of all sizes in the same group, and adjacent large vesicles tend to coalesce into irregular bullæ. Both the vesicles and the bullæ are frequently of irregular outline. Instead of being round or oval, as is usual with such lesions, they are elongated, angular, or stellate in outline, and in their stellate form may have a puckered appearance. The bullæ are usually tense in the beginning, with clear contents and with very little if any inflammatory areola, and vary in size from a pea to an egg. Later their contents become cloudy and areolæ develop. Around the bullæ are usually found vesicles. Neither the bullæ nor the vesicles are apt to rupture spontaneously. They rapidly develop to their full size with tense, distended walls, then their contents begin to be absorbed, the walls become flaccid, and ultimately the contents entirely disappear; the roof is exfoliated and a reddish stain remains, which vanishes leaving some pigmentation that is slow to disappear. The bullæ, until they begin to subside, are usually accompanied not so much by itching as by a very uncomfortable feeling of tension and heat. This disappears as the lesions become flaccid or when they are ruptured. Both the bullæ and the vesicles may become pustular from invasion of leucocytes without infection, but more frequently their contents become purulent by infection from without. In the bullous form of the disease the lesions present all sizes of vesicles and bullæ. The contents of the lesions may be serous at first, and become purulent later on, or they may be purulent from the start. The pustular lesions have a better defined inflammatory halo than those having serous contents. There may be in the pustular forms extravasation of blood into the lesions. The pustular lesions dry up, leaving yellowish or brownish crusts which are thrown off in the course of ten days to three weeks from the beginning of the lesions, leaving red and then pigmented stains. When the pustular type of lesions prevails the disease is usually of graver character with more constitutional disturbance than is the case with the other varieties.

The extent of the eruption in dermatitis herpetiformis varies greatly. There may be only a score or more of lesions widely distributed, or the lesions may be almost innumerable.

The eruption is bilateral and roughly symmetrical. It shows a predilection for the flexures and for the muco-cutaneous junctures, and is apt to be abundant on the face, in the axillæ and groins, around the genitals and anus and on the adjacent skin. It shows a certain predilection for the front of the trunk, especially for the abdomen, and for the buttocks. The distribution in some cases is limited to certain parts, as the axillæ, gluteal folds, and groins, or the wrists, elbows, and knees. It is common for the lesions to occur on the mucous membranes of the mouth and nose, less frequently of the eyes and pharynx, and occasionally of the trachea and larger bronchi. Lesions occurring upon the mucous membranes macerate and rupture quickly, leaving raw, unhealthy erosions over which a necrotic membrane may form. About the orifices of the body the lesions become covered with bloody crusts, erosions and fissures form, and thus most distressing conditions may be produced.

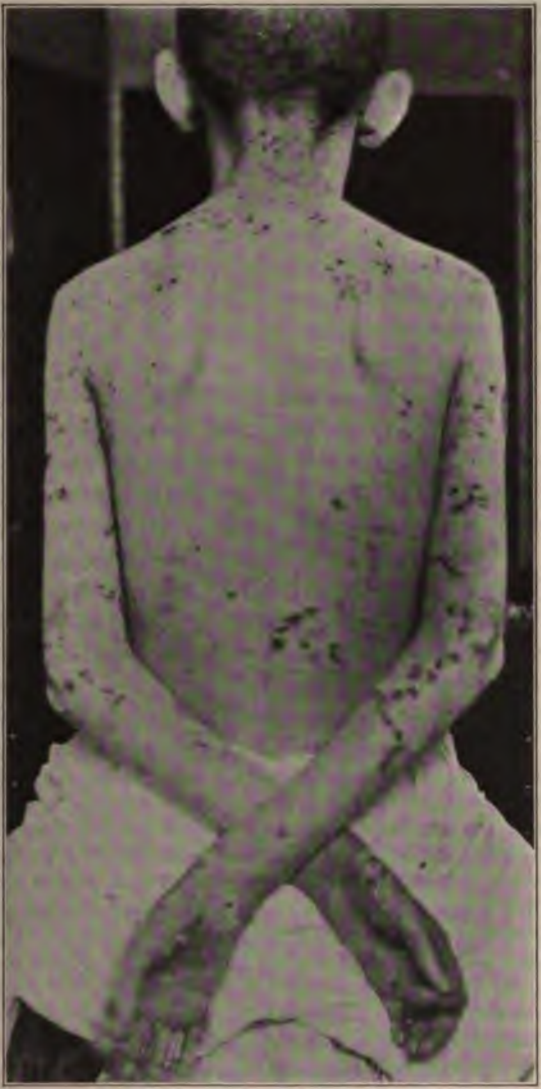

FIG. 55.—DERMATITIS HERPETIFORMIS. Vesiculo bullous type. (Author's collection.)

The arrangement of the lesions in groups has already been emphasized. In many cases this tendency to grouped arrangement is very marked; at times it is not so distinctive. The grouping usually takes the form of large central vesicles or bullæ with a cluster of smaller lesions around these. There may be one large bulla surrounded by satellite vesicles. In the grouping a circinate arrangement may be very well defined, and sometimes this amounts to the formation of rings of vesicles or of other lesions, with the production of iris forms. Hallopeau has described a case in which concentric rings of vesicles like rings of herpes iris occurred upon the trunk, and Wende and others have described similar cases.[1]

The lesions develop rather suddenly. They may appear within a few hours and attain their full development within twenty-four hours, but ordinarily their development is slower. They run a somewhat sluggish course, lasting from one to two or three weeks. The groups occur without any regularity, a group in one spot being followed by one in another, so that in a case lesions of all ages will be seen. The attacks are continued by the successive outbreaks of new crops of lesions. An outbreak of the eruption may last from two to four or five weeks, or much longer. Then it will subside and there will be a remission or an intermission, and after an uncertain interval a relapse will occur. The eruption in different relapses does not always show a uniform type. Sometimes this is the case, but in other cases one exacerbation will be of the vesicular type, another of a bullous or erythematous, or, rarely, pustular. In some cases the disease has shown great improvement as a result of intercurrent acute illnesses. The entire duration of the disease is variable. In fortunate cases it may get well after several months. In some of the cases it has lasted for years (five to fifteen years, Duhring's cases; six years, one of mine; ten years, one of Crocker's; twenty years, Brocq's). Finally the disease tends to disappear. The attacks become less frequent or less extensive and then cease altogether.

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[1] It is evident how closely iris groups of vesicles in dermatitis herpetiformis resemble the lesions of herpes iris; and this resemblance is interesting as tending to show how erythema multiforme and dermatitis herpetiformis in extreme cases may almost merge into each other. Indeed the two diseases in their pathogenesis and in their symptomatology show close resemblance. They are both vasomotor disturbances of bilateral distribution, and in both multiform lesions appear. Erythema multiforme, however, is a disease which runs a limited course, but it does occasionally pursue a chronic relapsing course; and so even in this respect extreme cases show their similarity to dermatitis herpetiformis. All of the various vasomotor disturbances can well be regarded as forming one group. Beginning with urticaria we have a vasomotor disturbance of the most ephemeral character which produces evanescent lesions; then we come to the hyperæmic erythemas which are vasomotor disturbances of slightly longer duration; then we have the erythema multiforme group of eruptions which usually pursue a course of a few weeks, but show an occasional tendency to relapse; and finally, we come to dermatitis herpetiformis and pemphigus, which are the gravest of the eruptions of vasomotor origin. In these we have persistent relapsing eruptions which, as a rule, are of more intense character than the other eruptions and are chronic in their course, because of a persistent or permanent vasomotor disturbance.

As already noted, the subjective symptoms accompanying the eruption of dermatitis herpetiformis are excessive. These symptoms are most frequently itching, or a sensation of tension and burning, and even pain. Itching is the characteristic sensation and it is especially marked in the erythematous, papular, and vesicular types of the disease, to a less extent in the pustular. In the bullous type the sensation is more frequently that of tension or burning or pain. Crocker refers to such a case, and I had a case in which there was no itching but a distressing sensation of tension and pain. The itching is apt to be most intense at night and when the lesions are developing.

As a result of itching there is scratching and occasionally secondary dermatitis with adenopathy and the usual phenomena of a scratched skin, but as a rule traumatism from scratching is not marked, and, considering the amount of distress which the patient has from the itching, the evidences of scratching are as a rule much less than would be expected.

The onset of the disease may be accompanied or preceded for a few days by some systemic disturbance, with chilliness and slight rise in temperature, and these symptoms may continue while the eruption is active. In many cases, however, constitutional symptoms are entirely lacking. As a result of the intolerable itching the patients are nervous, restless, and depressed, and suffer from insomnia, but the effect upon the general health is in most cases comparatively slight. The patients are frequently, however, distinctly below par both physically and nervously, but without any definite systemic disturbance. In the severest cases, especially in the pustular type, the general health may be much depressed; cachexia becomes marked, and ultimately the patients may develop chills, fever, diarrhœa, and pass into a typhoid state, perhaps become delirious, and die; or, sooner in the course, become the prey to pneumonia or some other intercurrent disease.

Complications.—In all of the forms of dermatitis herpetiformis the occurrence of urticarial wheals is not uncommon and petechiæ have been observed (Brocq, Tenneson). Papillomatous vegetations, like those of pemphigus vegetans, have been observed to develop upon excoriated surfaces (Brocq, Hallopeau, Wende, Crocker). These are probably analogous to the vegetating patches in eczema which have been recorded under vegetating dermatitis. The development of keratoses of the palms and soles, similar to those sometimes occurring in pemphigus, has been observed (Besnier, Brocq, Crocker). The disease has been observed to relapse into pemphigus (G. H. Fox), and Hallopeau recorded a case of long standing which developed into pemphigus foliaceus.

Etiology and Pathology.—The direct cause of dermatitis herpetiformis is unknown. Duhring in his earliest descriptions of the disease expressed his opinion that it was of nervous origin, and this is, I believe, certainly true in the majority of cases. Its onset is frequently a close sequence of some peculiar stress which has been thrown upon the nervous system; some severe mental or emotional strain or shock, such as failure in business, overwork, anxiety, grief, narrow escape from a serious accident. One form of the disease, herpes gestationis, is definitely associated with pregnancy. In some cases it has apparently been the result of a general infection. I[1] had one such case which followed quickly after vaccination, and was undoubtedly of internal origin and not due to local infection. But in this case in addition to the infection there were all of the factors of an unstable nervous temperament as a basis for the development of the disease. Similar cases following vaccination have been reported by Bowen and others. The disease occurs most frequently in the active period of adult life between twenty and forty. It has, however, been observed from three years to extreme old age. It is more frequent in men than in women.

The pathological process underlying the condition is a vasomotor disturbance, probably of central origin. As to what causes this disturbance we can only speculate. It may be direct irritation of the centers produced, as some of the French school believe, by the presence of toxins in the blood, or it may be the result of reflex irritation. Perhaps the presence of toxins explains the occurrence of the disease in certain cases, but in my opinion the disease in most cases is the result of vasomotor disturbance of purely nervous origin. No definite nervous lesions have been demonstrated in the disease.

As Leredde in particular has shown, there is a distinctive increase of eosinophile cells in this disease. This is noted in the blood, in the inflammatory exudate, and in the free serum in the vesicles and bullæ. In some cases the eosinophiles may be as low as four per cent of the white corpuscles; but they are nearly always from eight to fifteen per cent, and they may be as high as forty-seven per cent. This eosinophilia, according to Leredde, is greater than is usual in any other dermatoses except pemphigus vegetans and pemphigus foliaceus.

The urine shows no characteristic changes. Glycosuria, albuminuria, and decrease in urea and uric acid have been noted. According to Besnier, diminution in the quantity is usual, while on the other hand there may be polyuria. Engman,[2] in an examination of fourteen cases, has found indicanuria of marked degree a constant feature. With equal constancy there was eosinophilia, both conditions showing the greatest excess with exacerbation of the disease.

Most of the observers who have examined the lesions agree that the

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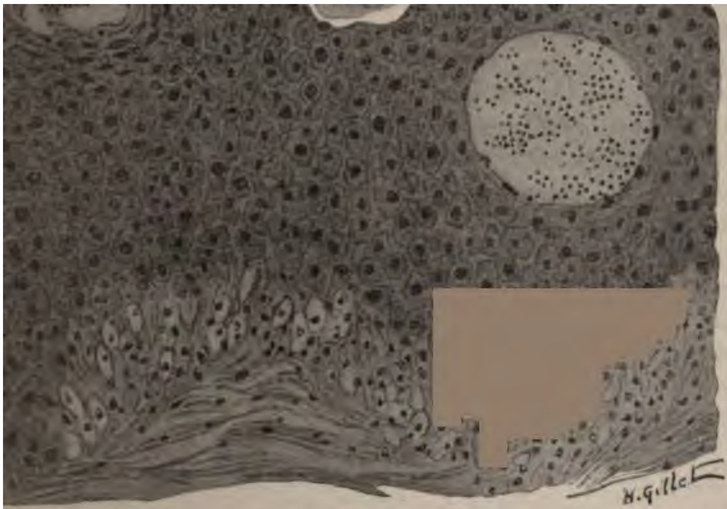
[1] *Jour. Cutan. Dis.*, April, 1897.

[2] Engman, *Jour. Cutan. Dis.*, May, 1906.



FORMATION OF VESICLE IN DERMATITIS HERPETIFORMIS. The vesicle on the has developed between the cells of the mucous layer. The cells surrounding flattened. The vesicle contains many cells, the majority being eosinophiles. esicle to the left has developed nearer the surface and is more advanced. It rounded by several layers of cells showing kerato-hyaline granules. A group sinophile cells is seen in the interior. (Leredde.)

leveloped within it interepithelial vesicles from the rupture of the œdema. The inflammatory changes in the corium are superficial, g chiefly the papillary and subpapillary portions. The changes e of an acute inflammatory process.

gnosis.—The occurrence of spontaneous multiform eruptions, ng some of the characteristics of urticaria, erythema multiforme, haps pemphigus, with extraordinary subjective sensations, and istory of repeated recurrences, suggests dermatitis herpetiformis.

erythema multiforme and of dermatitis herpetiformis is very similar, and there is little doubt that there are border-line cases in which the diagnosis is a matter of opinion. In urticaria all of the lesions have a definite wheal basis. The persistent forms of urticaria are, for the most part, characterized by papular lesions, and the disease does not show the multiformity of eruption which characterizes dermatitis herpetiformis. In urticaria, also, the lesions are due to vasomotor disturbances, and it is quite conceivable that certain vasomotor disturbances of profound origin which are characterized chiefly by wheals might be essentially of the same character as dermatitis herpetiformis. Clinically, however, urticaria is a sharply defined symptom complex which ordinarily presents no difficulty of differentiation from dermatitis herpetiformis.

The diagnosis from pemphigus will be considered under that disease.

Prognosis.—In no case of dermatitis herpetiformis can assurance be given of immunity from future outbreaks. The bullous and pustular types of the disease are the most unfavorable, and they may be followed by fatal termination, and are, perhaps, least liable to disappear. The usual vesicular and erythematous types tend in time to wear themselves out, and are likely to disappear entirely. These forms ordinarily show little effect upon the general health. The distress of an outbreak can be greatly relieved and attacks can usually be cured in a few weeks to a month, but recurrence is certain.

Treatment.—In the general treatment of dermatitis herpetiformis, usually the most important factor is the building up of the patient. He should be put in as favorable an environment as possible, and should be relieved as far as possible from demands upon his strength or his nervous energy. Abundance of rest and sleep, agreeable recreations, the avoidance of the taxes of vocation, a liberal diet, are all indicated. Tea, coffee, and tobacco in excess should be avoided, and alcohol altogether. During acute attacks rest in bed in an even temperature is an important therapeutic measure. In addition to these general measures it has been found empirically that arsenic, quinine, and strychnine in fairly full doses are very useful. If arsenic is used it should be pushed to its full physiological effect. Cod-liver oil is frequently useful, as are sometimes alkalies, diuretics, and aperients. Crocker recommends salicin in 15- to 30-grain dose, t.i.d. Sedatives are frequently demanded for the relief of itching. For this purpose phenacetin, antipyrin, the bromides, and belladonna may be used. Saline cathartics often are very useful in relieving itching.

For the relief of itching or other sensations which accompany the development of the lesions, puncture of the vesicles is the most efficient measure. With this may be combined the use of sedative lotions, such as calamine lotion, lotions containing one half to two per cent carbolic acid, thymol or camphor chloral, or five to ten per cent of one of the solutions of coal tar, like liquor picis alkalinus. In conjunction with these antipruritic lotions, and in less severe cases as substitutes for them, benefit can be gotten from the various soothing dusting powders. When the lesions become excoriated they should be protected by a bland ointment spread upon gauze. In some cases, then, general greasing of the surface with ointments is the most soothing measure. Duhring originally recommended the use of sulphur in the disease, and 10 to 30 grains of sulphur to the ounce is a useful addition to the ointments. In other cases for the relief of itching, tar in the proportion of 20 grains to 1 dram to the ounce is efficacious. The greatest relief is obtained by baths. For this purpose the best bath is usually a sulphur bath—2 to 4 ounces of potassium sulphide or of Vleminckx's solution to 30 gallons of water. Also useful are alkaline and bran baths. When the attacks are acute the repetition of these baths twice a day adds greatly to the comfort.

Herpes Gestationis

Herpes gestationis is dermatitis herpetiformis occurring in pregnancy.

It begins during pregnancy and usually recurs with each succeeding pregnancy. It may begin with the beginning of pregnancy, or later in its course, and is likely to continue until after delivery, when there is usually a violent outbreak of the disease, and it then subsides. Some cases beginning in pregnancy have persisted afterwards or recurred independently of it.

XII.—PEMPHIGUS[1]

Introductory

According to older usage pemphigus (from πέμφιξ, a blister) practically signified only that a disease had bullous lesions, and the term was applied to bullous eruptions of most widely different types. Martius, for example, with scholastic zeal enumerated ninety-seven varieties of pemphigus. The term is no longer used so loosely, and is now limited in its application to a definite disease or group of diseases which are characterized by the spontaneous development of bullæ.

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[1] Hebra, vol. ii, p. 361.—Kaposi, p. 390.—Zeisler, Morrow's "System."—Duhring, p. 449.—Brocq, *La Pratique*, iii, p. 723 (complete bibliography).—Grouven, *Archiv*, 1901, vol. lv, p. 85.—Audry, Gerard and Dalous, *Annales*, 1901, p. 113.—Unna, *Ther. d. Gegenw.*, 1901, No. 1.—Krxyztalowicz, *Monatshefte*, 1903, vol. xxxvi, p. 165.—Weidenfeld, Franz Deuticke, Wien, 1904.—Bronson, with discussion, *Jour. Cutan. Dis.*, March, 1906. MacLeod, *Brit. Jour. Dermat.*, 1909, p. 198.

There are many diseases in which bullæ occur that are not pemphigus. For example, they occur in burns, in infectious processes in the skin due to pus organisms, in congenital syphilis, in leprosy, rarely in urticaria and erythema multiforme. But none of these diseases is primarily bullous or resembles in hardly any particular the substantive disease which we know as pemphigus. In pemphigus the elementary eruption is bullous. The bullæ occur as result of an internal disturbance, they are the characteristic and primary eruption, and are not the sequelæ of any other eruption or of any local disturbance which can account for their origin. Modern usage limits the term pemphigus to the conditions which present these essential features.

It will serve to clear the ground if we first briefly refer to certain conditions which are still sometimes denominated as pemphigus, but which have little or no connection with it.

Pemphigus neonatorum, p. contagiosus, p. contagiosus tropicus, p. epidemicus. These are bullous forms of impetigo due to pustular infection of the skin.

P. syphiliticus is a bullous syphilide occurring in early congenital syphilis.

P. neuroticus is applied to the bullous eruption which not infrequently occurs in the course of many organic nervous diseases. It is seen with various irritative or paralytic nervous conditions, and most frequently in connection with diseases of the cord; in general paralysis, locomotor ataxia, myelitis, and progressive muscular atrophy. The pathological process which underlies it is a trophic disturbance due to interference with the trophic functions of the nerves innervating the affected part. Neurotic pemphigus approaches in its pathogenesis the true type of pemphigus.

P. leprosus is applied to the bullous eruption of leprosy which, unless traumatic, is of the same character as neurotic pemphigus.

P. hystericus [1] is applied to the very rare cases in which spontaneous bullæ occur as a complication of hysteria. It is related to neurotic pemphigus, but differs from it in that it is a vasomotor disturbance of emotional origin and not due to demonstrable lesions in the nervous system. The occurrence of bullæ in these cases is often associated with erythematous patches. The bullæ usually disappear by absorption without leaving scars, but in rare cases ulceration and gangrene have occurred in the lesions. A bulla, or a suppurating or a gangrenous ulcer in a hysterical person is always open to suspicion of self-infliction and usually has that origin, but there is good ground for believing that in very rare instances these lesions occur spontaneously as the result of the violent emotional disturbance of hysteria.

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[1] C. J. White, *Jour. Cutan. Dis.*, 1903.

Leaving these pemphigoid eruptions we now come to the conditions which are properly described as pemphigus.

Pemphigus is a disease, usually chronic, which is characterized by the primary development of crops of bullæ.

It is rare, constituting one to two *per mille* of skin diseases.

It occurs in four varieties—*p. vulgaris seu chronicus, p. acutus, p. foliaceus, p. vegetans.*

PEMPHIGUS CHRONICUS

This is the form of pemphigus which is understood when the term is used without qualification, and is the commonest variety of the disease.

Symptomatology.—The lesions of pemphigus are bullæ which develop rapidly, usually without discoverable preliminary erythema, and at first are tense, translucent, with thin walls, and without perceptible areolæ.

The bullæ are oval or roundish and vary in size from a pea to an egg or larger. Adjacent lesions tend to coalesce, and in this way large irregular bullæ may be formed. The contents of the bullæ are at first clear serum. This rapidly becomes, from invasion of leucocytes, milky and then puriform. Rarely there is sufficient admixture of blood with the serum to render the lesions hæmorrhagic, *p. hæmorrhagicus.*

The full development of the bullæ may take place within a few hours to twenty-four hours. After becoming fully developed, if unruptured by traumatism, their contents begin to disappear by absorption, a slight inflammatory areola is seen around each of them, they become flaccid, and in the course of about a week dry up. A single lesion runs its course in from one to two weeks. Upon the exfoliation of the epithelium forming the roof of the bulla, there is left a red stain and perhaps very slight pigmentation. If lesions develop repeatedly upon the same spot, pigmentation is more marked. If ruptured there is left a weeping surface over which horny epidermis quickly forms, leaving a red stain.

The eruption is, as a rule, irregularly bilateral. Only two cases are recorded (Pick, H. Neumann) in which it was unilateral.

There is no definite arrangement of the eruption. The lesions may come out singly or in crops at various points, and they may show some grouping, but not ordinarily the circinate arrangement which is seen in dermatitis herpetiformis. The disease may occur in crops of only one or two lesions at a time, which are likely to appear upon the extremities or the face, where the circulation is poor (*p. solitarius seu localis*), but the extent of the eruption varies from a very few lesions to great crops of bullæ.

The eruption may appear upon any part of the body, but shows a predilection for the lower part of the face and for the trunk and the extremities. In all forms of pemphigus lesions may occur upon the

mucous membranes. Their occurrence is usually an evidence o
form of the disease, and they are most common in pemphigus
It is a matter of diagnostic importance that the disease ma
first upon a mucous membrane, and indeed in some cases has a

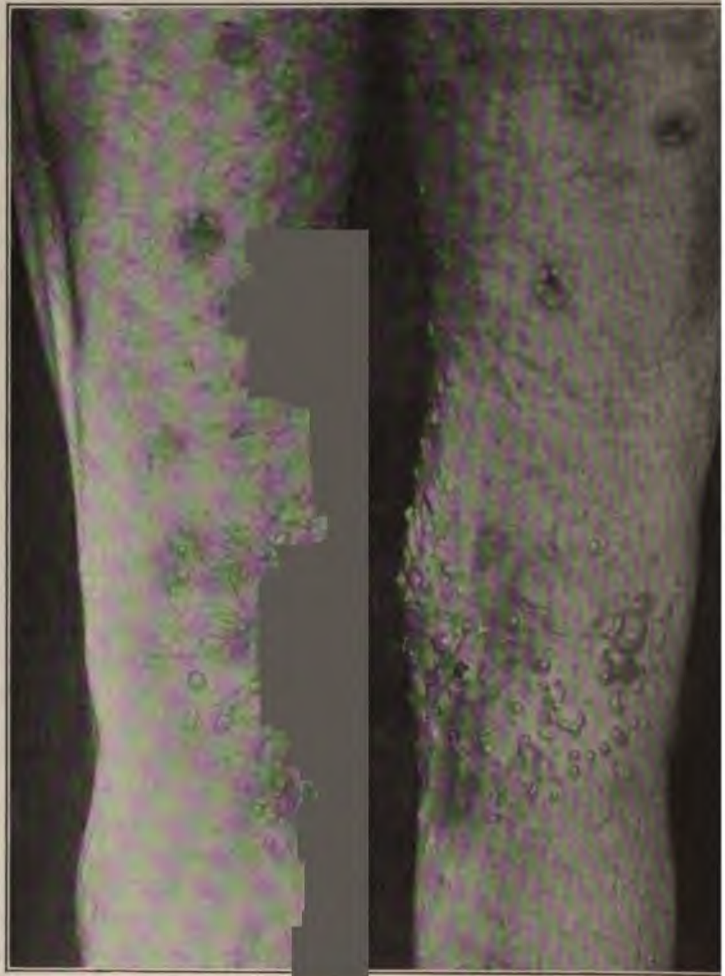

FIG. 57.—PEMPHIGUS. Lesions just developed, tense and clear without
(Author's collection.)

been confined to the mucous membrane of the mouth. The lesion
in the mouth as bullæ which rupture from maceration, usuall
they are detected, and leave excoriated surfaces which become
with a yellowish pellicle, and in the ordinary form of the diseas
heal over.

In addition to the mouth the lesions of pemphigus occur in the pharynx, larynx, nose, eyes, and vagina, and presumably the stomach. When the lesions occur upon adjacent mucous membranes adhesions are apt to form; such adhesions occasionally occur in the posterior nares, causing occlusions. When the lesions occur on the conjunctivæ adhesions may form between the ocular and palpebral surfaces. It is possible that the "essential shrinking of the conjunctivæ" of von Graefe is due to pemphigus, for in twenty-eight cases of the condition which Morris and Roberts[1] collected from the literature, sixteen had originally had pemphigus and others showed features suggestive of pemphigus.

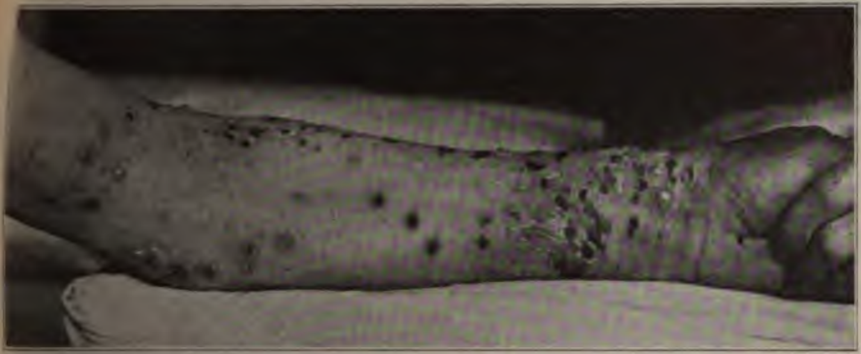

FIG. 58.—PEMPHIGUS. Same case as Fig. 57, lesions a few days older. (Author's collection.)

While the duration of an individual lesion will vary from one to two weeks, the disease is kept up by the irregular appearance of new lesions. These appear as individual lesions or as crops of lesions, and thus an attack will be continued for several weeks or several months. Usually new lesions then become fewer and fewer and the disease disappears. Only one attack of the disease may occur, but as rule it recurs after a few months or a year, and after several recurrences ceases altogether. In some cases the disease has been observed to be continuous, new lesions, it may be only a few, constantly appearing for many years or as long as the patient lived.

As a rule the subjective symptoms associated with the lesions are slight burning and tension, or even pain or itching as the lesions appear. The constitutional symptoms of pemphigus are usually slight, but vary with the abundance of the eruption. In mild cases they may be entirely absent. An outbreak of the disease may be associated with a slight fever and the usual accompanying symptoms. In children and old people, and when the eruption is abundant, the fever may amount to two or three degrees. Where extensive areas are denuded of epidermis or covered with bullæ, prostration is apt to be rather marked, with nervousness, insomnia, and anorexia. The constitutional disturbance continues, as a rule, as long as the disease is active. In severe, long-continued cases the prostration becomes marked, and death may occur from exhaustion or from some intercurrent disease.

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[1] *Brit. Jour. Derm.*, 1889, vol. i.

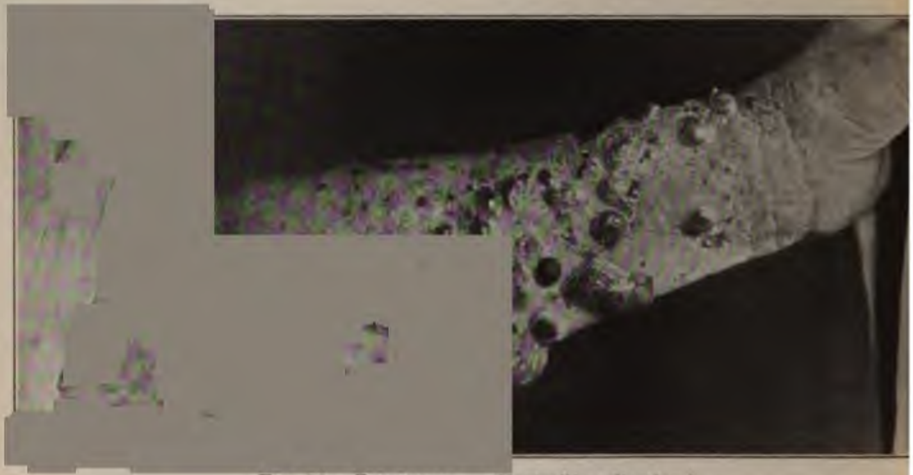

FIG. 59.—PEMPHIGUS. (Hartzell's collection.)

Complications.—Hyperkeratosis of the palms and soles may occur as a sequela of pemphigus, even when arsenic has not been given.

On the sites of the bullæ solid epidermal cysts are sometimes formed which look like milium bodies.

Crocker refers to a case in which convex erythematous swellings were left upon the disappearance of the bullæ.

Where the eruption is extensive, or when secondary dermatitis from scratching is well developed—an uncommon phenomenon in pemphigus—there may be lymphangitis with enlarged glands.

Variations.—Two of the variant forms of pemphigus, *p. foliaceus* and *p. vegetans*, are so definite as to merit separate descriptions. Other less characteristic variations need brief references. In very rare cases the excoriated bases of the bullæ become covered with a fibrinous exudate, *p. crouposus seu diphtheriticus*. In other cases the inflammatory process is so intense as to cause superficial or deep sloughing, *p. gangrænosus*, a form usually seen only in children. More or less admixture of blood may be present with the serum, producing bullæ which vary in color from pink to black, *p. hæmorrhagicus seu purpura bullosa.*

Under the heading of *p. circinatus* are described cases in which the eruption occurs as circinate groups of bullæ and vesicles. It is now regarded as a form of dermatitis herpetiformis, and does not differ in any essential from that disease.

Occasionally pemphigus is accompanied by intolerable itching, *p. pruriginosus*. The eruption appears as vesicles or small bullæ which are ruptured immediately by scratching, and the cases show all the features of the scratched skin. These cases are transitional forms between pemphigus and dermatitis herpetiformis, if indeed they are distinguishable from dermatitis herpetiformis.

PEMPHIGUS ACUTUS[1]

SYNONYMS: *Febris bullosa, P. febrilis.*

The term acute pemphigus is applied to cases which exhibit the cutaneous lesions of pemphigus, but differ in that they run an acute, usually febrile, short course, and are apt to terminate fatally. In recent years Pernet and others have described acute fatal cases which have occurred chiefly in butchers and others whose occupations expose them to septic animal poisons. In many of these cases a definite history of a preceding and apparently related wound has been obtained, and in most of them it would seem probable that the disease was due to some peculiar form

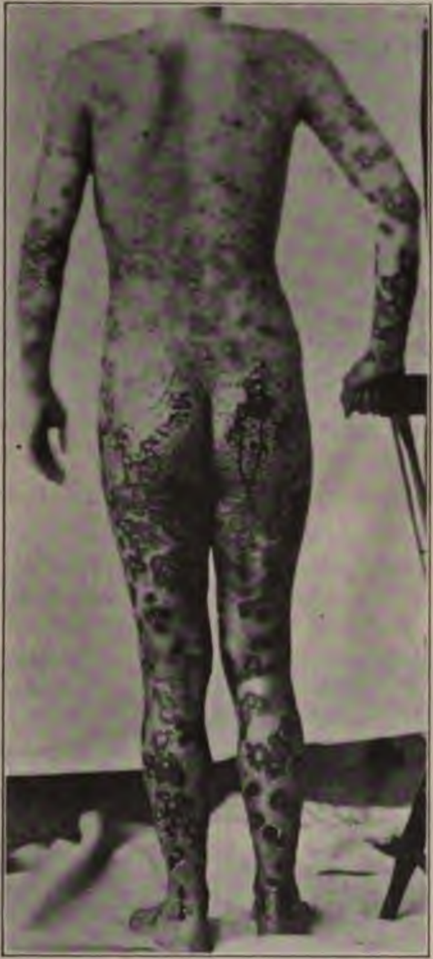

FIG. 60.—BULLOUS DERMATITIS FOLLOWING VACCINATION. ACUTE PEMPHIGUS. (Howe's photograph.)

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[1] Allen, *Jour. Cutan. Dis.*, vol. vi, 1888.—Pernet and Bulloch, *Brit. Jour. Derm.*, May, 1896 (full review).—Schamberg, *Annals of Gynecology and Pediatry*, February, 1901.—Howe, *Jour. Cutan. Dis.*, 1903 (report of ten cases).—Bowen, *Jour. Cutan. Dis.*, 1904 (review of subject); *Jour. Cutan. Dis.*, June, 1904, vol. xx; *Jour. Cutan. Dis.*, June, 1904.—Ormsby and Bassoe, *Jour. Cutan. Dis.*, July, 1905, p. 294.—Crary, *Jour. Cutan. Dis.*, January, 1906, p. 14.

of general sepsis. In the children's cases also the disease has seemed to be of toxic origin. It must be borne in mind, however, that in acute pemphigus the infection is a general infection, and the disease is not to

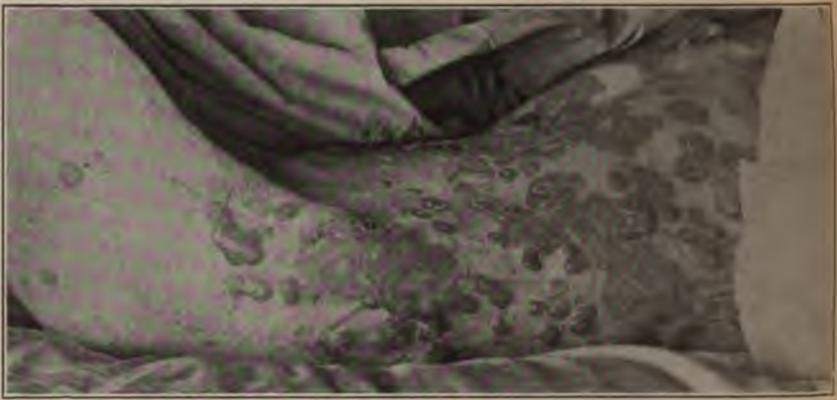

Fig. 61.—Acute Pemphigus in a Sheep Butcher. Pernet and Bulloch type. (Author's collection.)

be confused with the so-called epidemic pemphigus in children which is due to local infection with pus organisms. Bulloch found in the fluid of unruptured bullæ in Pernet's case and in Hardy's case a diplococcus

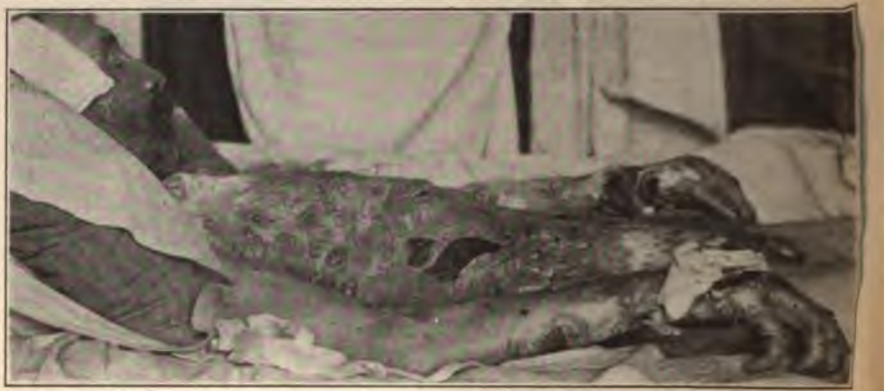

Fig. 62.—Acute Pemphigus. Same case as Fig. 61, two days later. The disease began in the large lesions on the backs of hands. (Author's collection.)

larger than the gonococcus, and a similar diplococcus was found by Demme and Bleibtreu in their cases.

A form of pemphigus resembling acute pemphigus of children, but running a somewhat more prolonged and favorable course, has been described by Hardy as *p. virginum* and by Tommasoli as *p. chloroticus.*

The disease occurred usually in chlorotic girls in the early years of adolescence, and was apparently of toxic origin.

PEMPHIGUS FOLIACEUS[1]

Pemphigus foliaceus presents the essential difference from ordinary pemphigus that the lesions, instead of healing rapidly, remain as excoriations which do not heal, or if they heal, heal imperfectly, leaving areas either denuded of epidermis or covered with unhealthy exfoliating epidermal scales.

Pemphigus foliaceus was originally described by Cazenave in 1844, and is a very rare affection.

The lesions of pemphigus foliaceus persist indefinitely, and as new bullæ are continually appearing the disease in time becomes universal. The entire surface, then, excepting perhaps the palms and soles, becomes involved in an exfoliative inflammatory process with an abundant discharge of sero-purulent secretion. This discharge dries on the surface in varnishlike crusts through which weeping fissures penetrate. The entire surface is thus marked off with irregular tessellated figures, which give it somewhat the appearance of the markings of large scales.

Pemphigus foliaceus may begin as an ordinary pemphigus or it may show its characteristic type from the start. The bullæ which develop are flaccid, and their contents accordingly collect in the dependent part. The contents of the bullæ are primarily purulent, or quickly become so. The bullæ rupture spontaneously, leaving excoriations which are surrounded by fringes of undermined epidermis under which the process spreads peripherally. Upon these denuded areas there may be an attempt at cornification, but it is imperfect and the scales either dry into crusts with the secretion or are thrown off with it. The amount of exfoliation in these cases is very abundant. Underneath the crusts the skin is red and weeping, and abortive bullæ are found developing here and there upon the unhealthy surface. The decomposition of the horn cells, added to that of the secretions, gives to these cases a peculiar, sweetish, nauseating odor.

Severe involvement of the mucous membranes is common in this form of pemphigus. As there is little tendency for regeneration of the epithelium, the excoriations in the mouth remain. The entire mouth may become involved, so that there is interference with swallowing and

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[1] Sherwell, *Jour. Cutan. Dis.*, vol. vii, 1889.—Hardaway, *Jour. Cutan. Dis.*, 1890.—Klotz, *Amer. Jour. Med. Sci.*, 1891.—Du Mesnil de Rochemont, *Arch. f. Derm. u. Syph.*, vol. xxx, 1895.—Biddle, *Jour. Cutan. Dis.*, 1897.—Fabry, *Arch. f. Derm. u. Syph.*, 1904, vol. lxx, p. 2.—Grinew, *Dermatol. Zeitschrift*, 1904, vol. xi, No. 12 (study of blood).

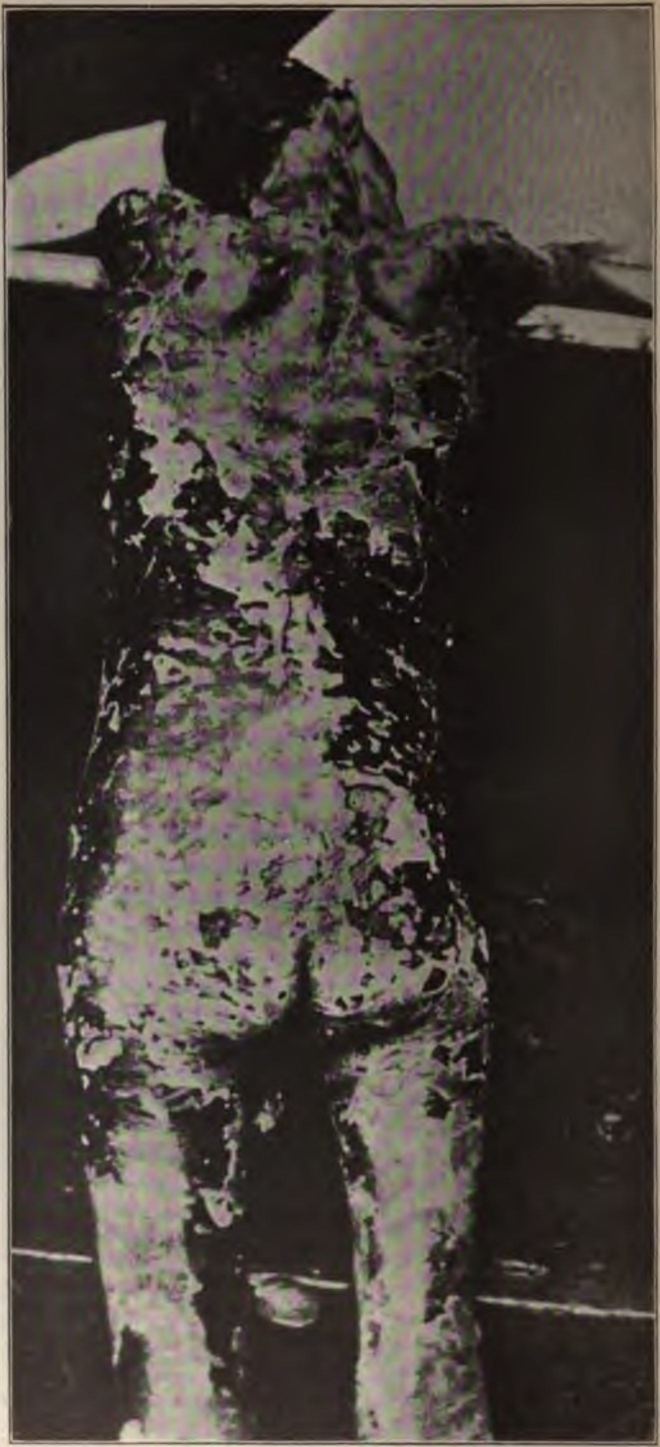

FIG. 63.—PEMPHIGUS FOLIACEUS, post mortem. (Lieberthal's photograph.)

even respiration. Such extensive involvement of the mouth is a late feature of fatal cases.

The nails become thinned, curved transversely and longitudinally, and may exfoliate. Much of the hair is lost, and the remnant is thin and short. The lids may become everted.

Itching is slight or absent in most cases, although occasionally it is severe. The surface, however, is sore and tender and stiff, with fissures around the joints, and the patients suffer considerable pain from contact with dressings and from the pressure of resting upon the parts. The disease is usually afebrile. It may continue for years with periods of improvement, but recovery rarely occurs. There is gradually increasing prostration, and the patients die from exhaustion or become the prey of some intercurrent disease like pneumonia. On the whole it is one of the most distressing of diseases.

PEMPHIGUS VEGETANS[1]

Pemphigus vegetans is a form of pemphigus in which the bullæ persist as excoriations upon which papillomatous vegetations develop.

It is an exceedingly rare disease. It was first described by Kaposi as *syphilis vegetans* in 1869, and by Auspitz as *herpes vegetans* in the same year. It was first recognized as a form of pemphigus by Neumann in 1886.

Symptomatology.—In many of the cases the first evidence of the disease has been pain or difficulty in swallowing, from the development of lesions upon the mucous membrane of the mouth or pharynx. It may begin, however, on any part of the body with bullous lesions indistinguishable from those of pemphigus. For a time these bullæ may run the usual course of pemphigus and the disease then lapse into pemphigus vegetans; or in other cases the bullæ from the start show the characteristics of pemphigus vegetans. The characteristic bulla of pemphigus vegetans leaves a base which does not heal, but remains as an excoriation which tends to spread at the periphery. When the lesions are exposed to warmth and moisture, as around the genitals and anus, the inguinal folds and the axillæ, there form upon the excoriated lesions papillomatous growths which closely resemble condylomata lata and secrete an offensive sero-purulent fluid. Some of the lesions of pemphigus vegetans may undergo involution, but others persist, and thus by the addition of new lesions the disease becomes very extensive. Vegetations may occasionally occur in any form of pemphigus. That which distinguishes true

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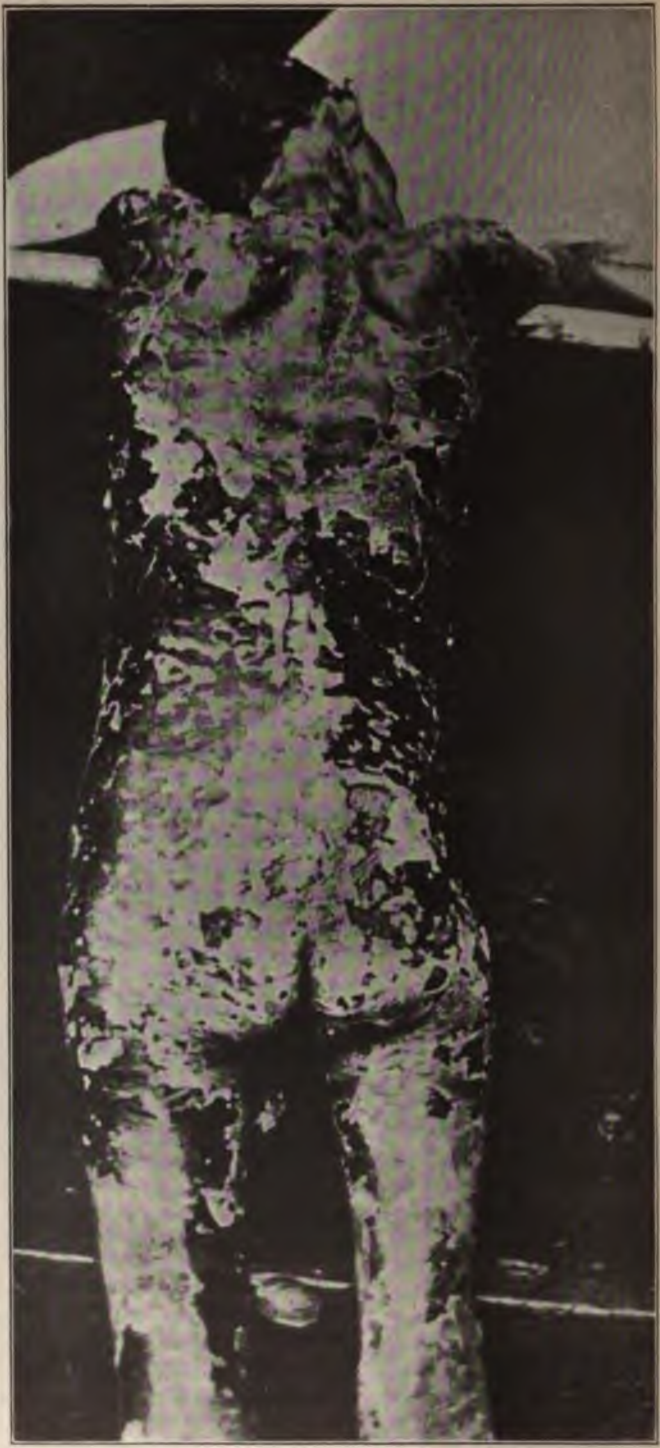
[1] Crocker, *Brit. Med. Jour.*, March 16, 1889; *London Med. Chi. Soc. Trans.*, vol. lxxii, 1889.—Hyde, *Jour. Cutan. Dis.*, vol. ix, 1891.—Weidenfeld, *Archiv*, vol. lxvii, p. 3.



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directly upon disturbances of the nervous system, and in other cases is the result of toxic conditions acting upon the vasomotor centers. In the case of acute pemphigus we come very near to demonstration that the cases are due to toxic disturbances, and the same is true in some of the cases of pemphigus foliaceus and pemphigus vegetans following infected wounds.

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In its pathogenesis pemphigus is, in my opinion, practically identical with dermatitis herpetiformis. Indeed, it seems likely that the two conditions are pathologically the same but present morphological differences, just, in fact, as different types of pemphigus and different types of dermatitis herpetiformis do.

The etiology of pemphigus foliaceus is unknown. The influence of chills as an exciting cause is recognized. It has been observed to follow violent emotional disturbances. One of the most interesting observations in connection with it is a case of Du Mesnil de Rochemont in which after an infection of the thumb it developed first on the corresponding arm and then spread over the whole body.

Most of the cases of pemphigus vegetans have occurred in women, and it is a disease of middle age. In a case of Haslund's it followed an infection of a finger, as de Rochemont's case of pemphigus foliaceus did. In Haslund's case, however, it first appeared in the mouth and on the vulva.

Pathology.—There is a divergence of opinion as to the method of formation of the pemphigus bulla. The inflammatory phenomena in the process are relatively slight, and many observers believe that the lesion is not the result of an inflammation, but is due to an œdema (a toxic œdema) causing, as Auspitz puts it, an acantholysis or rupture of the prickle-cell layer by the sudden invasion of fluid. Inflammatory phenomena, however, are not altogether lacking in the lesions of pemphigus even in the early stages, and *a priori* it would be expected that the lesions were due to an inflammatory process in which, as in urticaria, œdema was the most striking feature. That it is due to inflammation is the opinion of most authorities. In a careful examination of bullæ by Crocker there was free cellular infiltration of the upper layers of the corium, and Robinson found the corium infiltrated with leucocytes and with its blood vessels dilated. The bullæ may be formed in any part of the epidermis. Kaposi thought that the very superficial situation of the bullæ, just beneath the horny layer, was characteristic of pemphigus, but it has since been demonstrated that bullæ may occur in any part of the epidermis. They may be situated superficially between the horny layer and the stratum granulosum, or between the upper layers of the rete and the basal layer, or the entire epidermis may be raised and the bullæ form between the epidermis and the papillæ. The bullæ as an ordinary pemphigus and then lapse into pemphigus vegetans, that, even when well developed, ordinary lesions of pemphigus may occur, and the general resemblances to other forms of pemphigus leave little room for doubt that it is essentially a form of pemphigus, but, like pemphigus foliaceus, a form which fails to heal and which leaves persistent excoriations that are of a more destructive character than those even of pemphigus foliaceus. It is possible that the formation of vegetations is a secondary phenomenon due to inflammatory overgrowth from the constant presence of pus, warmth, and moisture, as has been observed in some cases of pemphigus foliaceus, dermatitis herpetiformis, and in vegetating dermatitis. I believe, however, from observation of an early case of pemphigus vegetans which I had the opportunity of studying, that this is not the proper explanation. In this case typical pemphigus bullæ were occurring, but on some of them which were not in the folds of the body and were not secreting pus, small early vegetations with spreading of the lesions at the border could be detected.

Etiology.—Nothing definite is known of the etiology of pemphigus. It occurs in both sexes indifferently and at all ages, although it is most frequent in children. There is no evidence of heredity as a causative factor and it is not contagious. It has been observed as a sequel of chilling of the surface with sufficient frequency to justify the belief that that is the exciting factor in some cases (Crocker, Kirschner). Crocker suggests that the pathogenesis of these cases may perhaps be found in increased absorption of toxins from the intestinal tract as result of the vascular dilatation which occurs in the reaction from the chills. The most definite factors in the etiology have to do with the nervous system. That the nervous system may produce the lesions of pemphigus is seen in pemphigus neuroticus and pemphigus hystericus, and similar eruptions have been repeatedly observed in connection with peripheral and central lesions of the nervous system. Aside, also, from any demonstrable lesions of the nervous system, many of the cases are associated with nervous strain, or breakdown, or emotional disturbance, in the same way that dermatitis herpetiformis is, and a study of such cases leaves little room for doubt that the nervous system is primarily at fault. That the nervous disturbances are functional in the sense that no demonstrable lesions can be found is well shown by the fact that of nine successive fatal cases examined *post mortem* by Kaposi, only one showed lesion of the nervous system, and that was a diffuse sclerosis of the cord. It occasionally develops from other diseases. It has been observed to develop from dermatitis herpetiformis (Hallopeau) and from the general exfoliative dermatitis of Wilson (Pringle).

Pemphigus is a vasomotor disturbance, a trophoneurosis, probably of central origin. It seems likely that in some cases it is dependent

directly upon disturbances of the nervous system, and in other cases is the result of toxic conditions acting upon the vasomotor centers. In the case of acute pemphigus we come very near to demonstration that the cases are due to toxic disturbances, and the same is true in some of the cases of pemphigus foliaceus and pemphigus vegetans following infected wounds.

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In the peculiar growths of pemphigus vegetans there is enormous proliferation of the rete with hypertrophy of the corresponding papillæ and inflammation of the corium. The lesions are, in other words, inflammatory acanthomata similar to condylomata.

The contents of the bullæ of pemphigus are serum and leucocytes. The contents are generally alkaline, but may be neutral or acid. Various microörganisms have been found in the bullæ. Staphylococci are usually present, streptococci occasionally. Gibier found small beaded micrococci in the bullæ and in the urine, and Crocker found micrococci in a recent bulla. But none of these findings are constant, and no essential relationship between the various organisms and the disease is at all likely. In the case of acute pemphigus the situation is different, for it seems quite possible that the diplococcus found by Demme and Bleibtreu is the pathogenic organism of the disease. In two cases of pemphigus vegetans Waelsch found pseudo-diphtheria bacilli which were fatal to rabbits and guinea pigs, but diphtheria antitoxin in one of his cases had no effect upon the lesions, except perhaps those in the mouth, and no pathogenic relationship of the organism to the disease could be proved.

Leredde has emphasized the importance of eosinophilia in pemphigus vegetans and pemphigus foliaceus as in dermatitis herpetiformis, and has tried to draw a distinction here from ordinary pemphigus, but eosinophilia is sometimes observed in pemphigus vulgaris and the distinction will not hold. Other blood findings are indefinite.

The urinary findings in all forms of pemphigus vary and are indefinite. They throw no light upon the disease and do not in any way substantiate the theories that pemphigus is due to defective urinary elimination, excess of ammonia in the blood, or any other definite metabolic disturbance.

Diagnosis.—The characteristic features of pemphigus are the spontaneous development of bullæ with either no antecedent lesions or at most a transient hyperæmia, the irregular development of these lesions either singly or in crops over a period varying from weeks to months, and the usual absence of subjective symptoms. Dermatitis herpetiformis differs from pemphigus in the grouping of the lesions, their tendency to circinate arrangement, their multiformity, and the intense itching or burning. In typical cases of either dermatitis herpetiformis or pemphigus there is no difficulty in making a diagnosis between the two. When, however, one comes to border-line cases between the two conditions the diagnosis becomes a matter of name. Indeed, the Vienna school, under the stimulus of the general acceptance which dermatitis herpetiformis has received, have broadened their description of pemphigus until they do not find it necessary to recognize such a disease as dermatitis herpetiformis. Duhring, however, holds to the distinction between the two conditions. As already stated, the probable truth is that dermatitis herpetiformis and pemphigus differ only morphologically. The great merit of Duhring's conception is that it has enlarged our view so that we can comprehend in one group all of these various but essentially related syndromes.

Other diseases in which bullæ occasionally occur, like erythema bullosum, urticaria bullosa, impetigo contagiosa, eczema, herpes, pompholyx, present individual characteristics which are so definite that they offer no difficulty of diagnosis from pemphigus. In epidermolysis bullosa the disease is congenital and the lesions can be produced by traumatism.

Acute pemphigus has to be diagnosticated from the bullous eruptions due to local infection, of which impetigo is the type. In these cases pus organisms are demonstrable, new lesions can be produced by inoculation the disease is infectious, and is likely to occur in epidemics. Bullou varicella occurs in epidemics and has a history of contagion, and run a short, self-limiting favorable course.

Pemphigus foliaceus is a universal exfoliative inflammatory diseas of the skin involving the mucous membrane of the mouth. It woul need to be diagnosticated from the few other universal dermatoses—ger eral exfoliative dermatitis of Wilson, pityriasis rubra of Hebra, pityrias rubra pilaris of Devergie, and universal eczema. In none of these di eases are there excoriations or bullous lesions upon the mucous men branes. All of them except universal eczema are dry, scaly eruption Eczema does not remain universal indefinitely, does not have the deve opment of flaccid purulent bullæ, is accompanied by severe itching, an is unaccompanied by the characteristic fetid odor and the progressi prostration of pemphigus foliaceus.

Pemphigus vegetans is very likely to be confused only with veg tating, papillomatous syphilidies—condylomata lata. Here the probab presence of syphilitic lesions in the mouth, the history of other syph litic eruptions, even if no other form of eruption be present, and tl prompt effect of treatment will clear the diagnosis.

Prognosis.—Pemphigus is a serious disease, and is often follow by death. Of unfavorable portent are abundance and long continuan of the eruption, advanced years, involvement of the mucous membrane systemic disturbances, especially chills, and purulent, hæmorrhagic, gangrenous lesions. Most cases of pemphigus vulgaris recover, but futu attacks are almost sure to occur. At times apparently mild cases b come continuous, and the disease in the rarest instances may lapse in pemphigus foliaceus.

Acute pemphigus is dangerous in proportion to the extent of tl eruption and the constitutional disturbance. The milder cases may get well; the severer cases die very quickly.

Patients with pemphigus foliaceus may live for many years, but the disease is in the end fatal. Sherwell has reported a recovery in which a girl had two typical attacks in successive years, and one atypical attack ten years later.

Well-developed pemphigus vegetans causes death in from three to twelve months, but in some atypical cases and some cases of limited extent and short course (as a case of Stopford Taylor's) recovery may occur.

Treatment.—The general management of pemphigus patients should be directed to supporting them and building them up in every way possible. Comfortable hygienic surroundings, change of scene and climate, a generous diet, and the use of tonics, are all strongly indicated. Among the medicinal tonics iron and cod-liver oil are frequently useful. Especially to be used, not only for their tonic effect but also for possible specific influence upon the disease, are arsenic, quinine, and strychnine. Arsenic is most highly regarded, but should be begun with moderate doses and pushed only gradually to the limit of toleration, and it should be given very cautiously if there are digestive disturbances or any other disturbances of the general health which it might possibly aggravate. In place of arsenic Crocker highly recommends salicin, 15 grains in water, t.i.d., increasing to 30 grains. Quinine should be given in full doses, as should strychnine, and Neisser advocates the hypodermic use of the latter. Pilocarpine and atropine are occasionally of service. Crocker cautions especially against the use of potassium iodide, because it generally exaggerates bullous eruptions and may make them gangrenous.

In acute pemphigus the treatment is wholly upon general principles, and is especially directed to supporting the patients in every way possible during the continuance of what is essentially a septic fever. In pemphigus foliaceus and pemphigus vegetans treatment is directed to meeting indications, and is generally along the same lines that are used in pemphigus vulgaris. Hutchinson especially recommends opium in pemphigus vegetans.

The local treatment of pemphigus consists in the application of means to soothe irritation and prevent infection. Itching cases are treated locally in the same way as dermatitis herpetiformis. The discomfort of bullæ is relieved by evacuating them. For the protection of the abraded surfaces bland antiseptic ointments, such as boric acid or weak carbolic-acid ointment, or liniments, such as calamine liniment, are useful. Where the disease is very extensive these should be spread upon cloths and the patients swathed in them. In pemphigus foliaceus the abundant use of mild antiseptic ointments or bland oils or liniments, like calamine liniment, serves to protect the surface and keep the patient comfortable. In all forms of pemphigus bland tepid baths are comforting, such as starch or gelatine or alkaline baths, or, best of all, sulphur baths. In Vienna the continuous bath at body temperature is used, and patients may live in such a bath for months in comfort, but I do not believe that on the whole the patients are as comfortable in continuous baths as they can be made in dressings. In all forms of pemphigus where the disease is very extensive the patients get great relief from the use of a water bed.

In pemphigus vegetans the greatest benefit is gotten from local antiseptic measures. Crocker recommends that the back and front of the body be covered with cloths soaked in two and one half per cent carbolic oil, the groins and axillæ dusted with an antiseptic powder, and the mouth frequently rinsed with dilute chlorinated soda and sprayed with dilute solution of potassium permanganate.

XIII.—IMPETIGO HERPETIFORMIS[1]

Impetigo herpetiformis is a disease characterized by the spontaneous development of an abundant eruption of closely grouped pinhead-sized pustules which are accompanied by grave systemic disturbance that usually ends fatally.

It is an excessively rare disease. It was first described by Hebra in 1872 from the study of five cases, and to his masterly description little has been added.

Impetigo herpetiformis presents striking similarities to dermatitis herpetiformis, and there has been much discussion as to whether it should be regarded as one form of that disease. In his first papers Duhring strongly advocated the position that it was a pustular form of dermatitis herpetiformis. It resembles dermatitis herpetiformis in the grouping of the lesions, their circinate arrangement, and their peripheral spreading. It differs in the typical cases such as Hebra first described in that the lesions are always minute pustules and are not multiform, in the absence of severe itching, in the severe systemic disturbance of apparently septic character, and in the generally fatal end. These differences are rather those between varieties of one pathological process than between diseases that are fundamentally different. All of these differences, too, are not sharply defined in all cases, for border-line cases with multiform lesions have been reported by Heitzmann, Zeisler, and Hartzell. The condition, to my mind, bears the closely analogous relation to dermatitis herpetiformis that acute cases of pemphigus of septic origin do to pemphigus chronicus.

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[1] Hebra, *Wien. med. Wochenschr.*, 48, 1872.—Heitzmann, *Arch. Derm.*, 1878.—Zeisler, *Monatshefte*, 1887.—Kaposi, *Viertelj. f. Derm. u. Syph.*, vol. xiv, 1887.—Hartzell, *Jour. Cut. and Gen.-Urin. Dis.*, 1897.—Sherwell, *Jour. Cut. and Gen.-Urin. Dis.*, 1889.—Whitehouse, *ibid.*, 1898.—Gunsett, *Arch. f. Derm. u. Syph.*, vol. lv, 1901 (full bibliography).

Symptomatology.—The eruption consists of minute pustules which develop upon an inflammatory base, usually in dense groups. The le- ns appear suddenly and are pustular from the start. They dry into lowish or yellowish-green crusts, and the disease extends at the periph- by the development of new pustules. Involved areas may remain nuded of epidermis or may heal, but the disease is continued by the

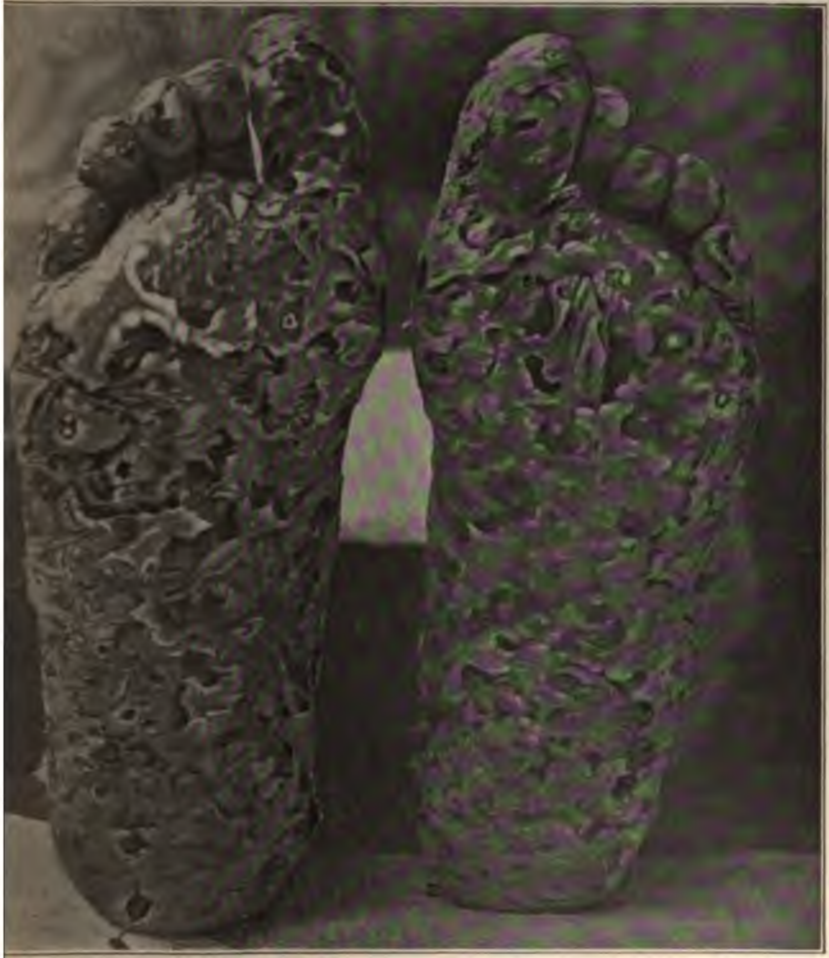

FIG. 65.—IMPETIGO HERPETIFORMIS. (Whitehouse's illustration.)

appearance of new lesions. The distribution is bilateral and roughly symmetrical, and the lesions show predilection for the genito-crural region, the inner surface of the thighs, the groins, the area around the navel, the breasts, the axillæ, and the mucous membrane of the mouth. Lesions are found not only in the mouth, but in the nose and pharynx, and Kaposi found them *post mortem* in the œsophagus. Occasionally the lesions are discrete, but there is a marked tendency to development in dense, crowded groups, and to arrangement in circles. As the eruption spreads by the development of new lesions at the periphery, adjacent groups become confluent, and thus the tendency to the formation of circinate and gyrate figures is in many cases very marked. The disease may become almost or quite universal, and it usually involves large areas of skin. The involved areas are covered with yellowish or greenish crusts, under which the skin is inflamed and denuded of epidermis, like a weeping eczema. On the diseased areas new lesions spring up, and, where they show a strong tendency to circinate arrangement, they may mark the areas with most striking circinate figures, which Whitehouse compares to marquetry. The eruption appears irregularly or in crops. There may be exacerbations and remissions, but the gradual tendency is downward.

Itching or other subjective symptoms accompanying the eruption are slight or absent. The onset of the disease and the outbreak of new crops of eruption are accompanied by chills and slight rise in temperature, which in Whitehouse's case showed regular morning remissions and evening exacerbations to 102.5° F. or less. There is great and increasing prostration, frequently with diarrhœa, and the patients die apparently of a slow sepsis. There has been usually high-colored urine with increase of urea but without albuminuria.

Etiology and Pathology.—Less than thirty cases have been described. Over two thirds of these were in Vienna and only three in America. Four fifths of the cases have been in women, and in most of the female cases the disease has occurred in association with pregnancy. The disease has supervened upon other inflammatory diseases of the skin. Whitehouse's and Breier's cases were preceded by eczema, one of Kaposi's by a severe intertrigo of the genito-crural region, and Rille's by an iodoform dermatitis. The association of the disease in some cases with purulent infection of the skin, its occurrence in pregnancy, its development with rigors and fever, and especially the close resemblance of the long-continued constitutional disturbance to general sepsis indicate strongly an infectious cause of the disease, and there is nearly unanimity in the view that it is a disease due to general infection.

Histologically there is an inflammatory process involving the corium and epidermis. Numerous microörganisms have been found in the lesions, but without any evidence of a causal relationship.

Diagnosis.—The diagnostic features of the disease are the small size of the pustules and their pustular character from the start, their marked tendency to grouping, usually in circinate groups, and to peripheral spreading, the grave systemic disturbance accompanying the disease, and its occurrence in a large majority of cases in pregnant women. The disease is most likely to be confused with pustular dermatitis herpetiformis. The differences between the two conditions have been referred to above. When it has become universal it may be confused with pemphigus foliaceus, but from all forms of pemphigus it is distinguished by the entire absence of bullæ.

Prognosis.—The disease in almost all cases runs a fatal course in from a few weeks to a year or more. In Gunsett's case, which became almost universal, the patient recovered under the administration of quinine.

Treatment.—The treatment is purely symptomatic. The local treatment is similar to that of pemphigus.

XIV.—EPIDERMOLYSIS BULLOSA[1]

SYNONYM: *Acantholysis bullosa.*

Epidermolysis bullosa is an hereditary condition of the skin in which bullæ form from slightest mechanical traumatism.

It is very rare, and was first described by Goldscheider in 1882.

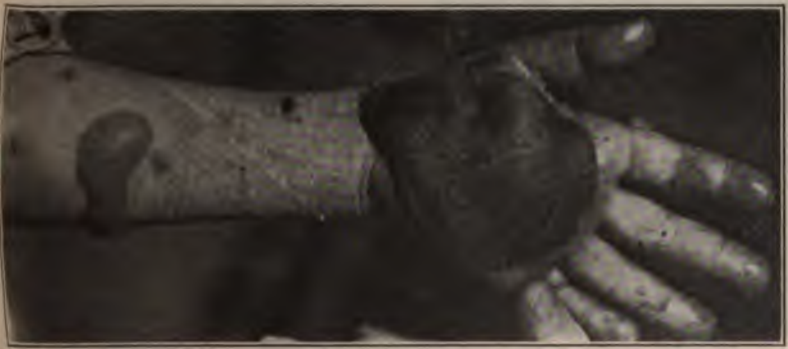

FIG. 66.—EPIDERMOLYSIS BULLOSA. Hæmorrhagic lesion on palm from fall; bulla on arm produced by friction. (Engman and Mook's collection.)

Symptomatology.—Epidermolysis bullosa consists essentially of an abnormal vulnerability of the skin, so that mechanical insults that would

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[1] Beatty, *Brit. Jour. Derm.*, vol. ix, 1897 (full review).—Bettman, *Arch. f. Derm. u. Syph.*, vol. lv, 1901.—Elliot, *Jour. Cut. and Gen.-Urin. Dis.*, January, 1895.—Wende, *Jour. Cut. and Gen.-Urin. Dis.*, 1902.—Bowen, *Jour. Cut. and Gen.-Urin.*

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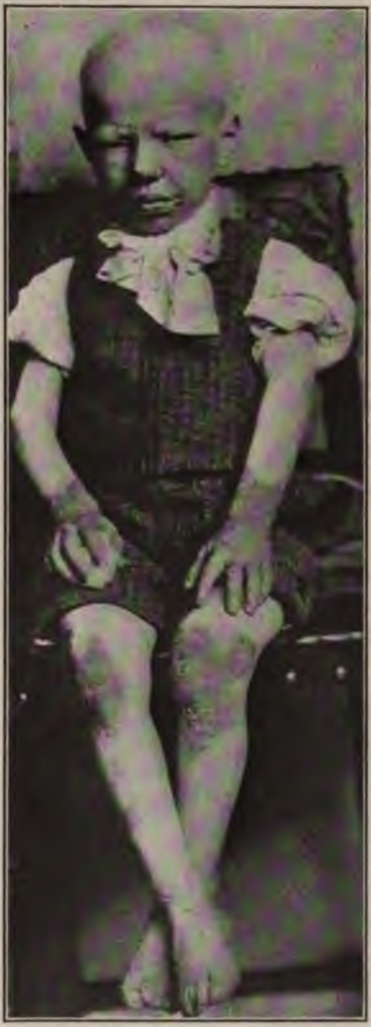

FIG. 67.—EPIDERMOLYSIS BULLOSA. (Grover W. Wende's collection.)

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the lesions occur chiefly u
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tion, on the hands and fe
the bony prominences, and w
skin is most exposed to the pressure and friction of clothing
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Dis., 1898.—Smith, H. L., *Amer. Med. Jour.*, April, 1901 (case in a
Weidenfeld, "Beiträge zur Klinik und Pathogenese des Pemphigus," Vien
—Engman and Mook, *Jour. Cutan. Dis.*, February, 1906 (histology an
raphy).

formation of bullæ, there is often atrophy and deformity of the fingers with deformity and loss of the nails. Thinning of the skin also and freckling have been observed at points where no bullæ have occurred (Bettman). In a case quoted by Wende there was alopecia. In cases of Startin's and Crocker's the condition was associated with another congenital defect of the skin, ichthyosis. As in pemphigus, milium bodies often develop upon the sites of lesions.

Etiology and Pathology.—The condition is congenital and hereditary. It has been traced through four (Bettman) and five (Bonaiuto) generations. It usually begins with the first month of life, and is said to cease between forty and fifty, but it has been observed to persist at fifty-five and sixty-four. Williams has reported a case indicating its existence *in utero*. A condition analogous to that of hereditary epidermolysis bullosa has been observed to develop after pemphigus. In a case of Colcott Fox's the patient, a woman aged fifty-one, after the disappearance of a pemphigus which had existed for nine years, developed the same susceptibility to external injuries that is seen in epidermolysis bullosa. The case is interesting in connection with Weidenfeld's observation that in some cases of pemphigus bullæ can be produced by pressure. The condition is essentially an abnormal vulnerability of the

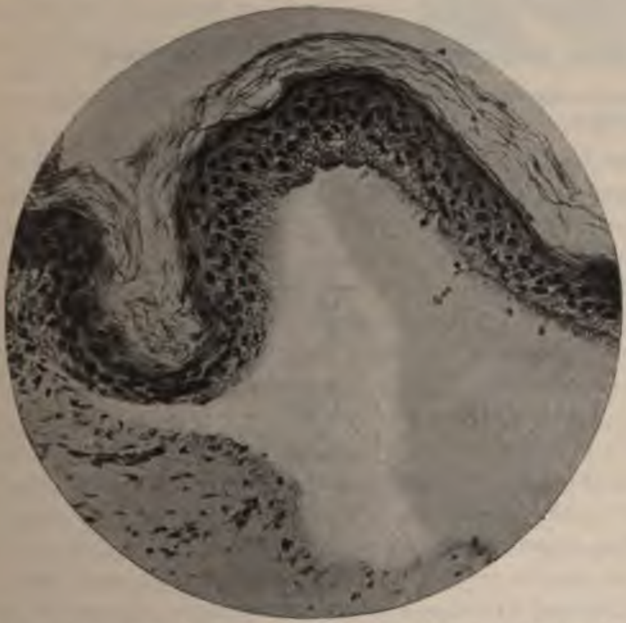

FIG. 68.—EPIDERMOLYSIS BULLOSA. Section of bulla twelve hours old. Degeneration of basal layers can be seen. (Elliot.)

directly upon disturbances of the nervous system, and in other cases is the result of toxic conditions acting upon the vasomotor centers. In the case of acute pemphigus we come very near to demonstration that the cases are due to toxic disturbances, and the same is true in some of the cases of pemphigus foliaceus and pemphigus vegetans following infected wounds.

In its pathogenesis pemphigus is, in my opinion, practically identical with dermatitis herpetiformis. Indeed, it seems likely that the two conditions are pathologically the same but present morphological differences, just, in fact, as different types of pemphigus and different types of dermatitis herpetiformis do.

The etiology of pemphigus foliaceus is unknown. The influence of chills as an exciting cause is recognized. It has been observed to follow violent emotional disturbances. One of the most interesting observations in connection with it is a case of Du Mesnil de Rochemont in which after an infection of the thumb it developed first on the corresponding arm and then spread over the whole body.

Most of the cases of pemphigus vegetans have occurred in women, and it is a disease of middle age. In a case of Haslund's it followed an infection of a finger, as de Rochemont's case of pemphigus foliaceus did. In Haslund's case, however, it first appeared in the mouth and on the vulva.

Pathology.—There is a divergence of opinion as to the method of formation of the pemphigus bulla. The inflammatory phenomena in the process are relatively slight, and many observers believe that the lesion is not the result of an inflammation, but is due to an œdema (a toxic œdema) causing, as Auspitz puts it, an acantholysis or rupture of the prickle-cell layer by the sudden invasion of fluid. Inflammatory phenomena, however, are not altogether lacking in the lesions of pemphigus even in the early stages, and *a priori* it would be expected that the lesions were due to an inflammatory process in which, as in urticaria, œdema was the most striking feature. That it is due to inflammation is the opinion of most authorities. In a careful examination of bullæ by Crocker there was free cellular infiltration of the upper layers of the corium, and Robinson found the corium infiltrated with leucocytes and with its blood vessels dilated. The bullæ may be formed in any part of the epidermis. Kaposi thought that the very superficial situation of the bullæ, just beneath the horny layer, was characteristic of pemphigus, but it has since been demonstrated that bullæ may occur in any part of the epidermis. They may be situated superficially between the horny layer and the stratum granulosum, or between the upper layers of the rete and the basal layer, or the entire epidermis may be raised and the bullæ form between the epidermis and the papillæ. The bullæ within twenty-four hours or less, and then begins involution. The contents of the vesicle may be absorbed, leaving a crust, or the vesicle may rupture with the formation of a crust upon the excoriation, or, most characteristic, the center of the vesicle sinks down into a thin reddish crust around which there persists a vesicular ring which may spread somewhat at the periphery. In this stage there is a close resemblance to a vaccine vesicle. The crust finally forms over the entire vesicle and exfoliation takes place. The peculiar feature of the lesions is that after exfoliation of the crusts there are usually left atrophic reddish scars which ultimately become white and look like the scars of vaccinia. The active lesions last for three to four days; the crusts exfoliate in the course of about a week more. The lesions develop without any regularity as regards time, so that lesions in all stages of their course will be found during an attack, and an attack will be prolonged for two or three weeks. The distribution is a characteristic feature. The eruption appears symmetrically, and sparsely scattered lesions may occur all over the body, but there is a strong predilection for exposed surfaces—the cheeks, nose, ears, the neck (especially the sides), the back of the hands, and, less frequently, the extensor surfaces of the arms, forearms, and legs. As the lesions are followed by slight scarring, and as they recur from year to year upon the same sites, it is evident that very considerable disfigurement in time may be produced. This shows most markedly in the atrophic scarring which ultimately occurs upon the ears, the nose, and the fingers, and to a somewhat less extent upon the cheeks.

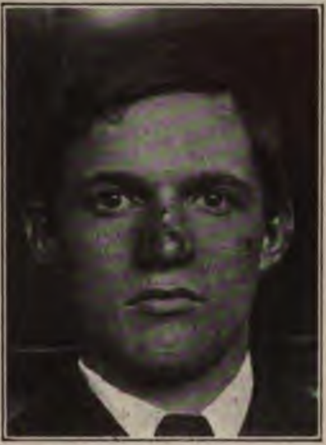

FIG. 69.—HYDROA VACCINIFORME. (Author's collection.)

An attack may be preceded by slight indisposition, but the constitutional symptoms are usually trivial. Before the appearance of the eruption there is a sensation of burning, or pain, or tension, which persists until the lesions become fully developed. Itching in most cases is slight, but occasionally is pronounced. The history of the disease as a whole is one of repeated recurrences. The ordinary type is excited by heat, and in such cases recurrences are likely to take place at any time from spring to fall. Similar recurrences are apt to develop throughout the winter in the winter type of the disease.

Etiology and Pathology.—The disease is by far most frequent in boys, but occurs in girls. It usually begins in the second or third year of life, but has been observed to develop as late as thirteen, eighteen, they do not find it necessary to recognize such a disease as dermatitis herpetiformis. Duhring, however, holds to the distinction between the two conditions. As already stated, the probable truth is that dermatitis herpetiformis and pemphigus differ only morphologically. The great merit of Duhring's conception is that it has enlarged our view so that we can comprehend in one group all of these various but essentially related syndromes.

Other diseases in which bullæ occasionally occur, like erythema bullosum, urticaria bullosa, impetigo contagiosa, eczema, herpes, pompholyx, present individual characteristics which are so definite that they offer no difficulty of diagnosis from pemphigus. In epidermolysis bullosa the disease is congenital and the lesions can be produced by traumatism.

Acute pemphigus has to be diagnosticated from the bullous eruptions due to local infection, of which impetigo is the type. In these cases pus organisms are demonstrable, new lesions can be produced by inoculation, the disease is infectious, and is likely to occur in epidemics. Bullous varicella occurs in epidemics and has a history of contagion, and runs a short, self-limiting favorable course.

Pemphigus foliaceus is a universal exfoliative inflammatory disease of the skin involving the mucous membrane of the mouth. It would need to be diagnosticated from the few other universal dermatoses—general exfoliative dermatitis of Wilson, pityriasis rubra of Hebra, pityriasis rubra pilaris of Devergie, and universal eczema. In none of these diseases are there excoriations or bullous lesions upon the mucous membranes. All of them except universal eczema are dry, scaly eruptions. Eczema does not remain universal indefinitely, does not have the development of flaccid purulent bullæ, is accompanied by severe itching, and is unaccompanied by the characteristic fetid odor and the progressive prostration of pemphigus foliaceus.

Pemphigus vegetans is very likely to be confused only with vegetating, papillomatous syphilidies—condylomata lata. Here the probable presence of syphilitic lesions in the mouth, the history of other syphilitic eruptions, even if no other form of eruption be present, and the prompt effect of treatment will clear the diagnosis.

Prognosis.—Pemphigus is a serious disease, and is often followed by death. Of unfavorable portent are abundance and long continuance of the eruption, advanced years, involvement of the mucous membranes, systemic disturbances, especially chills, and purulent, hæmorrhagic, or gangrenous lesions. Most cases of pemphigus vulgaris recover, but future attacks are almost sure to occur. At times apparently mild cases become continuous, and the disease in the rarest instances may lapse into pemphigus foliaceus.

Acute pemphigus is dangerous in proportion to the extent of the eruption and the constitutional disturbance. The milder cases may get well; the severer cases die very quickly.

Patients with pemphigus foliaceus may live for many years, but the disease is in the end fatal. Sherwell has reported a recovery in which a girl had two typical attacks in successive years, and one atypical attack ten years later.

Well-developed pemphigus vegetans causes death in from three to twelve months, but in some atypical cases and some cases of limited extent and short course (as a case of Stopford Taylor's) recovery may occur.

Treatment.—The general management of pemphigus patients should be directed to supporting them and building them up in every way possible. Comfortable hygienic surroundings, change of scene and climate, a generous diet, and the use of tonics, are all strongly indicated. Among the medicinal tonics iron and cod-liver oil are frequently useful. Especially to be used, not only for their tonic effect but also for possible specific influence upon the disease, are arsenic, quinine, and strychnine. Arsenic is most highly regarded, but should be begun with moderate doses and pushed only gradually to the limit of toleration, and it should be given very cautiously if there are digestive disturbances or any other disturbances of the general health which it might possibly aggravate. In place of arsenic Crocker highly recommends salicin, 15 grains in water, t.i.d., increasing to 30 grains. Quinine should be given in full doses, as should strychnine, and Neisser advocates the hypodermic use of the latter. Pilocarpine and atropine are occasionally of service. Crocker cautions especially against the use of potassium iodide, because it generally exaggerates bullous eruptions and may make them gangrenous.

In acute pemphigus the treatment is wholly upon general principles, and is especially directed to supporting the patients in every way possible during the continuance of what is essentially a septic fever. In pemphigus foliaceus and pemphigus vegetans treatment is directed to meeting indications, and is generally along the same lines that are used in pemphigus vulgaris. Hutchinson especially recommends opium in pemphigus vegetans.

The local treatment of pemphigus consists in the application of means to soothe irritation and prevent infection. Itching cases are treated locally in the same way as dermatitis herpetiformis. The discomfort of bullæ is relieved by evacuating them. For the protection of the abraded surfaces bland antiseptic ointments, such as boric acid or weak carbolic-acid ointment, or liniments, such as calamine liniment, are useful. Where the disease is very extensive these should be spread upon cloths and the patients swathed in them. In pemphigus foliaceus the abundant use of mild antiseptic ointments or bland oils or liniments,

SECTION IV

INFLAMMATIONS[1]

In this group are included, for the most part, such forms of dermatitis as are, or may be, excited by external irritants. Drug eruptions are included here, because it is not desirable to separate them from dermatoses produced by other chemical irritants, although from the internal use of drugs almost all forms of eruptions are seen.

I.—ECZEMA

SYNONYMS: *Tetter, Salt Rheum.*

Introductory.—Eczema is the commonest disease of the skin. The clinical pictures which it presents are usually sharp and clear, and experts have no difficulty in agreeing on the diagnosis in any given case of ordinary type. But what eczema is, and what it is not, what constitutes eczema, and what is a proper definition of it, are questions upon which there is considerable divergence of statement. These questions are by no means of purely academic interest, for a fundamental necessity for a satisfactory conception of any subject is a clear idea of what it is. For this reason it seems advisable to consider this topic at the outset.

Hebra first cleared the subject of eczema of some of its vagueness, and also opened the discussion as to its proper definition, by declaring that eczema is dermatitis such as can be excited in any skin by external irritants. And most authors agree, or at least admit incidentally in their descriptions of eczema, that its eruption is indistinguishable from dermatitis; for example, Stelwagon says, "Inflammation of the skin of artificial origin is often similar in symptomatology to that of eczema and may, indeed, be considered identical." But many authorities are not willing to rest with this. They insist that it differs from an ordinary dermatitis of external origin by certain added factors, usually factors of personal equation in the patient. The position of Malcolm Morris ith multiform lesions have been reported by Heitzmann, Zeisler, and lartzell. The condition, to my mind, bears the closely analogous rela- on to dermatitis herpetiformis that acute cases of pemphigus of septic igin do to pemphigus chronicus.

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[1] Fordyce, "The Modern Conception of Eczema," *Jour. A. M. A.*, June 13, 1903. —Bowen, "Modern Theories and Treatment of Eczema," Boston, *Med. and Sur. Jour.*, October 10, 1895.—Bulkley, "Eczema and Its Management," G. P. Putnam's Sons.—Bulkley, "Neurotic Eczema," *Med. News*, January 31 and February 7, 1891. —Dockrell, "Research into the Alleged Parasitic Nature of Eczema."

Symptomatology.—The eruption consists of minute pustules which velop upon an inflammatory base, usually in dense groups. The le- s appear suddenly and are pustular from the start. They dry into wish or yellowish-green crusts, and the disease extends at the periph- by the development of new pustules. Involved areas may remain ded of epidermis or may heal, but the disease is continued by the

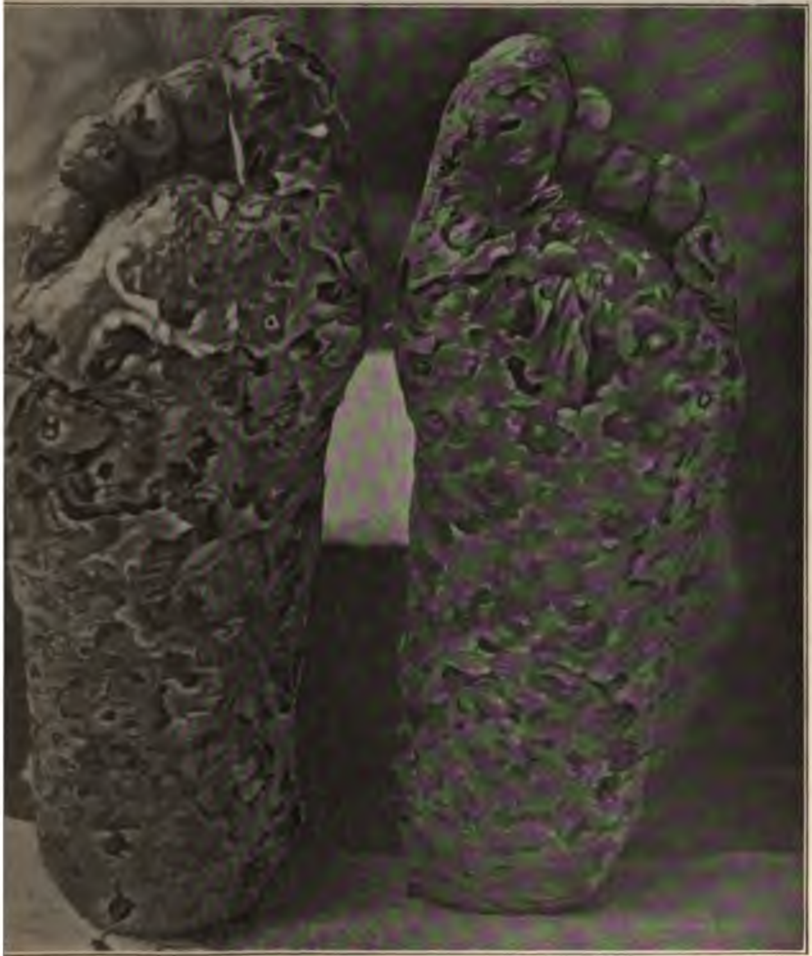


Fig. 65.—Impetigo Herpetiformis. (Whitehouse's illustration.)

appearance of new lesions. The distribution is bilateral and roughly symmetrical, and the lesions show predilection for the genito-crural region, the inner surface of the thighs, the groins, the area around the navel, the breasts, the axillæ, and the mucous membrane of the mouth. Lesions are found not only in the mouth, but in the nose and pharynx, and Kaposi found them *post mortem* in the œsophagus. Occasionally the lesions are discrete, but there is a marked tendency to development in dense, crowded groups, and to arrangement in circles. As the eruption spreads by the development of new lesions at the periphery, adjacent groups become confluent, and thus the tendency to the formation of circinate and gyrate figures is in many cases very marked. The disease may become almost or quite universal, and it usually involves large areas of skin. The involved areas are covered with yellowish or greenish crusts, under which the skin is inflamed and denuded of epidermis, like a weeping eczema. On the diseased areas new lesions spring up, and, where they show a strong tendency to circinate arrangement, they may mark the areas with most striking circinate figures, which Whitehouse compares to marquetry. The eruption appears irregularly or in crops. There may be exacerbations and remissions, but the gradual tendency is downward.

Itching or other subjective symptoms accompanying the eruption are slight or absent. The onset of the disease and the outbreak of new crops of eruption are accompanied by chills and slight rise in temperature, which in Whitehouse's case showed regular morning remissions and evening exacerbations to 102.5° F. or less. There is great and increasing prostration, frequently with diarrhœa, and the patients die apparently of a slow sepsis. There has been usually high-colored urine with increase of urea but without albuminuria.

Etiology and Pathology.—Less than thirty cases have been described. Over two thirds of these were in Vienna and only three in America. Four fifths of the cases have been in women, and in most of the female cases the disease has occurred in association with pregnancy. The disease has supervened upon other inflammatory diseases of the skin. Whitehouse's and Breier's cases were preceded by eczema, one of Kaposi's by a severe intertrigo of the genito-crural region, and Rille's by an iodoform dermatitis. The association of the disease in some cases with purulent infection of the skin, its occurrence in pregnancy, its development with rigors and fever, and especially the close resemblance of the long-continued constitutional disturbance to general sepsis indicate strongly an infectious cause of the disease, and there is nearly unanimity in the view that it is a disease due to general infection.

Histologically there is an inflammatory process involving the corium and epidermis. Numerous microörganisms have been found in the lesions, but without any evidence of a causal relationship.

Diagnosis.—The diagnostic features of the disease are the small ize of the pustules and their pustular character from the start, their arked tendency to grouping, usually in circinate groups, and to periphral spreading, the grave systemic disturbance accompanying the disease, nd its occurrence in a large majority of cases in pregnant women. The isease is most likely to be confused with pustular dermatitis herpetirmis. The differences between the two conditions have been referred above. When it has become universal it may be confused with pemigus foliaceus, but from all forms of pemphigus it is distinguished the entire absence of bullæ.

Prognosis.—The disease in almost all cases runs a fatal course in om a few weeks to a year or more. In Gunsett's case, which became most universal, the patient recovered under the administration of quine.

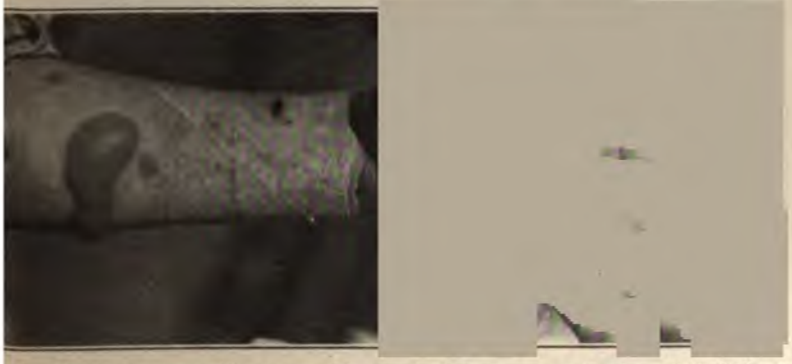
Treatment.—The treatment is purely symptomatic. The local eatment is similar to that of pemphigus.

XIV.—EPIDERMOLYSIS BULLOSA[1]

Synonym: *Acantholysis bullosa.*

Epidermolysis bullosa is an hereditary condition of the skin in which læ form from slightest mechanical traumatism.

It is very rare, and was first described by Goldscheider in 1882.



66.—Epidermolysis Bullosa. Hæmorrhagic lesion on palm from fall; bulla on arm produced by friction. (Engman and Mook's collection.)

Symptomatology.—Epidermolysis bullosa consists essentially of an ormal vulnerability of the skin, so that mechanical insults that would

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[1] Beatty, *Brit. Jour. Derm.*, vol. ix, 1897 (full review).—Bettman, *Arch. f. Derm. ph.*, vol. lv, 1901.—Elliot, *Jour. Cut. and Gen.-Urin. Dis.*, January, 1895.—de, *Jour. Cut. and Gen.-Urin. Dis.*, 1902.—Bowen, *Jour. Cut. and Gen.-Urin.*

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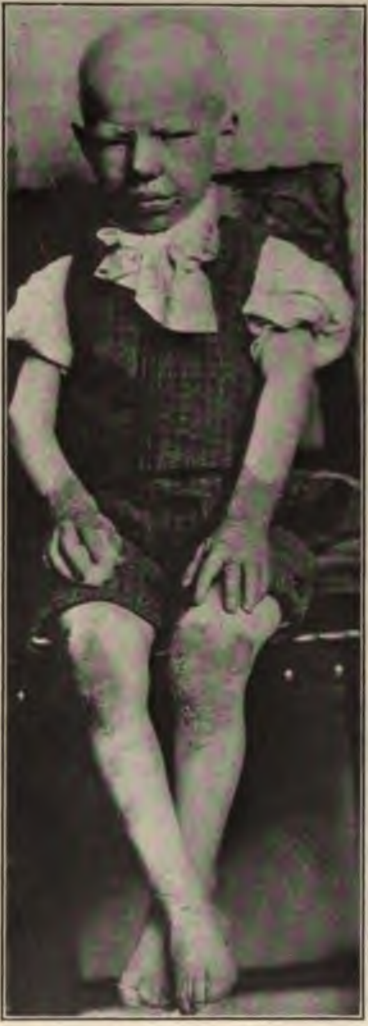

FIG. 67.—EPIDERMOLYSIS BULLOSA. (Grover W. Wende's collection.)

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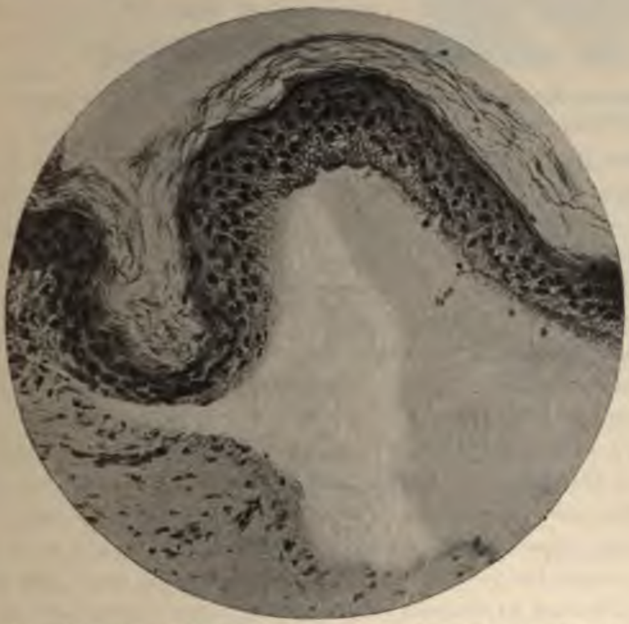

FIG. 68.—EPIDERMOLYSIS BULLOSA. Section of bulla twelve hours old. Degeneration of basal layers can be seen. (Elliot.)

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on that in some cases of pemphigus bullæ can be produced by pres-
. The condition is essentially an abnormal vulnerability of the

epidermis. The abnormal vulnerability of the skin in different cases of epidermolysis bullosa varies greatly. In some cases it is so extreme as to almost completely incapacitate the individual.

As Elliot has shown, the lesions are accompanied by a true inflammatory process in the corium with sudden and violent outpouring of serum which macerates and causes degeneration of the rete cells and ruptures the rete in its lower part with the formation of bullæ.

Engman and Mook have discovered the absence of elastic fibers in cases of epidermolysis bullosa. The elastic tissue is absent in the papillary and subpapillary regions of the derma and sparsely distributed and deformed in the deeper regions.

Treatment.—Treatment is of no avail against the condition. All that can be done is the avoidance as far as possible of the exciting causes. The local treatment consists of puncture of the bullæ and the use of protective antiseptic applications until healing takes place.

XV.—HYDROA[1] VACCINIFORME[2]

SYNONYMS: *Hydroa æstivale*; *Recurrent summer eruption* (Hutchinson); *Hydroa puerorum* (Unna).

Hydroa vacciniforme is a recurrent inflammatory dermatosis, excited by extremes of temperature and characterized by the appearance upon the exposed parts of a vesicular eruption which upon disappearing leaves scars.

The disease is a rare one which was first described by Bazin in 1862 and independently by Hutchinson in 1888.

Symptomatology.—It begins usually during the second or third year of life and consists essentially of an unusual vulnerability to such sources of irritation as arise from exposure to the weather. The exciting causes are usually exposure to sunlight or heat and to winds. Occasionally the irritability is manifested to cold. The disease develops upon exposed parts and recurs with the seasons. The eruption begins as red macules upon which tense round vesicles quickly develop, either singly or in herpetic groups. In size the vesicles vary from a pinhead to a small pea, and adjacent vesicles tend to coalesce into irregular bullæ surrounded by inflammatory areolæ. The contents of the vesicles are at first serous and then become milky or perhaps puriform. A vesicle reaches its development

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[1] Hydroa (from ὕδωρ, water) was a name formerly given to many bullous eruptions. The only disease in which the name survives is hydroa vacciniforme.

[2] White, Jas. C., *Jour. Cut. and Gen.-Urin. Dis.*, vol. xvi, 1898; *Trans. Ame Derm. Assn.*, June 1, 1898.—Anderson, *Brit. Jour. Derm.*, January, 1898.—Bowe *Jour. Cut. and Gen.-Urin. Dis.*, vol. xii, 1894 (bibliography and histology).—Mibell *Monat.*, vol. xxiv, 1897 (bibliography).—Fox, C., *Brit. Jour. Derm.*, vol. x, 1898.—Adamson, *Brit. Jour. Derm.*, April, 1906 (review and bibliography).

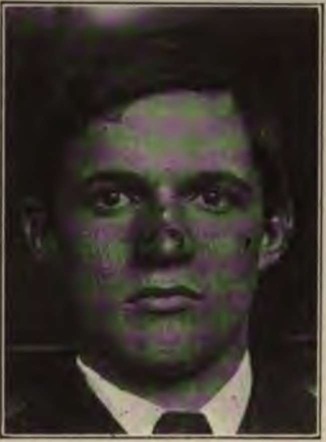

FIG. 69.—HYDROA VACCINIFORME. (Author's collection.)

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and twenty-six years. Most of the late cases have been in women. Th summer type of the disease is distinctly excited by heat— by the heat o the sun and by artificial heat as well; these cases also show frequentl a susceptibility to irritation by wind and are excited by exposure t wind. The exciting cause is apparently heat and not, as in xeroderm pigmentosum, the actinic rays of light, for exposure to direct heat whe the actinic rays are minimal, as artificial heat, excites the condition and protection of the parts against light by covering with substance opaque to the actinic rays has apparently had no effect. J. C. White ha reported two cases which were exaggerated in winter, in which the exciting factors were cold and winter winds. These cases became quiescent in summer. Colcott Fox also has reported a winter case, so tha we must revise our conception of it as a summer disease and regard i rather as a disease which is excited by the irritating factors of th weather of either of the extremes of season.

It is a vasomotor disturbance arising from undue susceptibility t certain forms of external irritation. This susceptibility is usually congenital, but in some of the cases developing later in life it has apparentl been acquired. In three cases reported by White there was eosinophilia of from eight to fifteen per cent. In two cases of McCall Anderson's the urine was a Burgundy red color from the presence of a pigmen allied to urohematoporphyrin, but the urinary findings generally hav not been significant. Anatomically it is an inflammatory process in th upper part of the corium and in the rete.

Hydroa vacciniforme in its morphology and in the pathological mechanism of its eruption resembles more closely simple herpes than it doe the graver bullous diseases, like dermatitis herpetiformis and pemphigu These are due to vasomotor disturbances of central origin, while hydro vacciniforme is excited by local irritation. The underlying factor i its production is a personal predisposition which consists of unusua vulnerability of the skin to certain forms of external irritants. In thi respect there is an analogy with epidermolysis bullosa and dermographism, both of which consist of an unusual vulnerability of the ski to another form of external irritation, viz., mechanical irritation. further analogy between hydroa vacciniforme and epidermolysis bullos is seen in the disfigurement of the fingers which occurs in repeated attacks of hydroa vacciniforme and in the disfigurement of the finger which results from repeated lesions of epidermolysis bullosa.

Diagnosis.—The diagnostic features of hydroa vacciniforme are vesicular eruption which is followed by scarring, symmetrical distribution on exposed parts, and distinct association with extremes of temperature; the beginning of the disease in most cases in childhood; and th recurrences from year to year in the same season. These features ar sufficient to diagnosticate it from any of the other diseases which it superficially resembles, as erythema multiforme, dermatitis herpetiformis, xeroderma pigmentosum, eczema, and the diseases which are followed by superficial scarring, lupus vulgaris, lupus erythematosus, acne varioliformis, syphilis.

Treatment.—Avoidance of the exciting causes is, of course, most important. Internally arsenic may be useful, or salicin or quinine. With the hope of avoiding scarring the vesicles should be punctured and antiseptic powders like iodoform or aristol, or an ointment containing such antiseptics, should be applied to the surface. Where crusts form they should be softened and removed with two per cent carbolized oil or ointment, and the excoriations dressed with antiseptic ointments. In some cases ichthyol and resorcin, in the form of pastes and ointments, have seemed beneficial.

SECTION IV

INFLAMMATIONS [1]

In this group are included, for the most part, such forms of dermatitis as are, or may be, excited by external irritants. Drug eruptions are included here, because it is not desirable to separate them from dermatoses produced by other chemical irritants, although from the internal use of drugs almost all forms of eruptions are seen.

I.—ECZEMA

SYNONYMS: *Tetter, Salt Rheum.*

Introductory.—Eczema is the commonest disease of the skin. The clinical pictures which it presents are usually sharp and clear, and experts have no difficulty in agreeing on the diagnosis in any given case of ordinary type. But what eczema is, and what it is not, what constitutes eczema, and what is a proper definition of it, are questions upon which there is considerable divergence of statement. These questions are by no means of purely academic interest, for a fundamental necessity for a satisfactory conception of any subject is a clear idea of what it is. For this reason it seems advisable to consider this topic at the outset.

Hebra first cleared the subject of eczema of some of its vagueness, and also opened the discussion as to its proper definition, by declaring that eczema is dermatitis such as can be excited in any skin by external irritants. And most authors agree, or at least admit incidentally in their descriptions of eczema, that its eruption is indistinguishable from dermatitis; for example, Stelwagon says, "Inflammation of the skin of artificial origin is often similar in symptomatology to that of eczema and may, indeed, be considered identical." But many authorities are not willing to rest with this. They insist that it differs from an ordinary dermatitis of external origin by certain added factors, usually factors of personal equation in the patient. The position of Malcolm Morris may be cited as an example of the point of view of those who hold that eczema is something more than a dermatitis. Morris, than whom no one is better able to state a case, gives the following definition of eczema: "Eczema is . . . a catarrhal inflammation of the skin originating without visible external irritation and characterized in some stage of its evolution by serous exudation. . . . The definition of eczema here given excludes all forms of inflammation of the skin caused by chemical or mechanical irritants. The artificial dermatitis set up by such agents is *identical anatomically with the eczematous process* and gives rise to *lesions indistinguishable from those of eczema,*[1] but it is not eczema." This seems to me a *reductio ad absurdum.* It amounts to saying that two things which are the same are different because they are produced differently. The most that this statement can mean is that there is dermatitis of external origin and dermatitis of other origin which are indistinguishable, and that he would confine the conception of eczema to the dermatitis of other origin. Morris goes on: "It is evident, therefore, that there is something more in eczema than inflammation of the skin due to a local and transient cause. There is an unknown quantity beyond this, a pathological x which may be either some invisible source of irritation or some constitutional peculiarity or both of these factors." These identical words could be used in favor of the position that pneumonia and tuberculosis are not essentially due to the pneumococcus and the tubercle bacillus respectively. Indeed, in the old days this line of argument was used to prove that tuberculosis was a "constitutional" disease. If it were not for this pathological x, some factor of lowered resistance, we would all have pneumonia all the time; and who of us would escape tuberculosis? Further, his qualification that the process originates without *visible* external irritation is also certainly an unessential condition. It makes no difference in the essential character of a dermatitis that the irritant happens in some cases to be discoverable and in others not.

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[1] Fordyce, "The Modern Conception of Eczema," *Jour. A. M. A.*, June 13, 1903. —Bowen, "Modern Theories and Treatment of Eczema," Boston, *Med. and Sur. Jour.*, October 10, 1895.—Bulkley, "Eczema and Its Management," G. P. Putnam's Sons.—Bulkley, "Neurotic Eczema," *Med. News*, January 31 and February 7, 1891. —Dockrell, "Research into the Alleged Parasitic Nature of Eczema."

Leaving out these unessential qualifications we cannot avoid the position that eczema is dermatitis; and, as far as the lesions in the skin are concerned, this statement represents the fact. The eruption of eczema is the same as that of dermatitis, and as the only essential feature of eczema is its eruption, we are forced to the definition that the term eczema connotes various forms of simple dermatitis. By the qualification "simple" is meant that the process is such as can be excited by ordinary external irritants. For example, erythema multiforme, herpes zoster, psoriasis, and various other dermatoses are inflammatory processes, but they are not forms of simple dermatitis that can be reproduced by ordinary irritants, and their eruptions do not come within our conception of eczema. Usage has further limited the term eczema to certain forms of dermatitis. A violent dermatitis with bullæ, such as is produced by a superficial burn or by an intense reaction from croton oil or poison ivy, or a dermatitis which is but a part of a deep cellulitis, does not come within the accepted conception which usage has given to eczema; further, most circumscribed forms of dermatitis produced by drugs or other external irritants of definite character are by usage described as dermatitis. But these are matters of convention—justified in most instances by convenience in describing briefly clinical differences—and have nothing to do with the essential identity of the processes. According to this conception, eczema is not a substantive disease, it is not a distinct pathological entity; it is rather a group of symptoms in the skin which can be produced by innumerable causes. And this is the fact.

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[1] The italics are mine, W. A. P.

This conception of eczema does not necessitate that every inflam matory process in the skin be regarded as an eczema. We can eliminat from our conception of eczema all definite forms of dermatitis as rap idly as we discover that they are definite, just as gonorrhœa was sepa rated from other forms of urethritis when its specific character wa determined. We have already eliminated certain diseases, as, for e ample, seborrhœic dermatitis and scabies, in the one case chiefly becau of the characteristic and definite clinical picture which the disease pr sents, and in the other because we know that the dermatitis is a sequel a pathological condition produced by the invasion of the skin by a speci parasite. And there is no doubt that this elimination of definite diseas from the large group of affections which we now regard simply eczema will go further. In other words, as our knowledge becomes full eczema becomes more and more circumscribed.[1] It is impossible, my opinion, ever to separate a disease, to which we will confine the ter eczema, from all the forms of dermatitis to which the name eczen has so long been applied. The tendency of scientific classification in the opposite direction, and as soon as a definite clinical form dermatitis is established in the group of eruptions that we now ca eczema it is taken out of the group and given an independent pla in nosology.

The view that eczema cannot be distinguished from dermatitis is n original here. All authors practically agree that the lesions are indi tinguishable; and most of those who are unwilling to cast themselves away from the conception of eczema as a substantive disease are forced to admit tacitly or by inference that the eruptions are in many cases indistinguishable. Crocker states the matter clearly, as follows: "I believe, therefore, that it is more logical and practical not to draw such arbitrary distinction (between dermatitis and eczema, W. A. P.), and to consider all cases as eczema which correspond in their morphology and general behavior irrespective of the cause being tangible or intangible, external or internal."

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[1] This conception of eczema is the old conception of Hebra. The effort to g away from it has been due to the attempts to see in eczema a definite disease. Th effort reached its climax in Unna's contention that eczema was due to a defini organism—his morococcus—and probably its decline will be dated from the Inte national Dermatological Congress of Paris in 1900 when Unna's morococcus fell fl

In the description which follows—and no departure is made from the accepted description of eczema—we shall be simply defining various forms of dermatitis.

Symptomatology.—When an inflammatory reaction occurs in a spot in the skin there is produced first a red macule from hyperæmia; if the inflammatory process increases, the swelling from the exudate forms a red papule; if the process goes still further, the inflammatory œdema invading the epidermis ruptures the epidermis and collects in a cavity, forming a vesicle on a hyperæmic base. The contents of this vesicle may become purulent from invasion and breaking down of leucocytes, forming a pustule, or the roof of the vesicle may rupture and leave a blood-red point denuded of horny epidermis and therefore weeping. Let us assume that the inflammatory process in our lesion becomes less acute but does not disappear; as a result there will be a proliferation of connective-tissue cells with the formation of a chronic inflammatory induration. The spot then will be indurated and thickened and covered by a layer of epidermis which, as a result of the subacute inflammatory process, proliferates more rapidly than normal, but does not form perfect horny scales and consequently exfoliates more rapidly than normal; in other words, we have a scaling hyperplastic spot. These changes in our hypothetical spot of inflammation in the skin represent all of the types of eczema—erythematous, papular, vesicular, pustular, weeping, and squamous. It is the innumerable combinations of these various inflammatory lesions which produce the pictures of dermatitis which constitutes eczema.

Eczema is divided into four chief varieties, erythematous, papular, vesicular, and pustular. This division into varieties is in part arbitrary, for the purpose of convenience of description. It is rare for any picture of eczema to be of uniform type, and if it is of uniform type it usually does not remain long without the development of lesions of another type; as the process varies in intensity the lesions change.

Erythematous Eczema.—Erythematous eczema consists of areas of dry dermatitis. The skin is pink to reddish in color, the redness disappearing on pressure; there is slight scaliness of the surface, which gives it a ground-glass appearance; and the borders are ordina defined and gradually merge into healthy skin. The swelling is slight, but in lax tissues, like the eyelids, it may be pronounce condition varies in intensity, and is very apt to be complicated presence over its of a greater or le ber of papules, and weeping poin

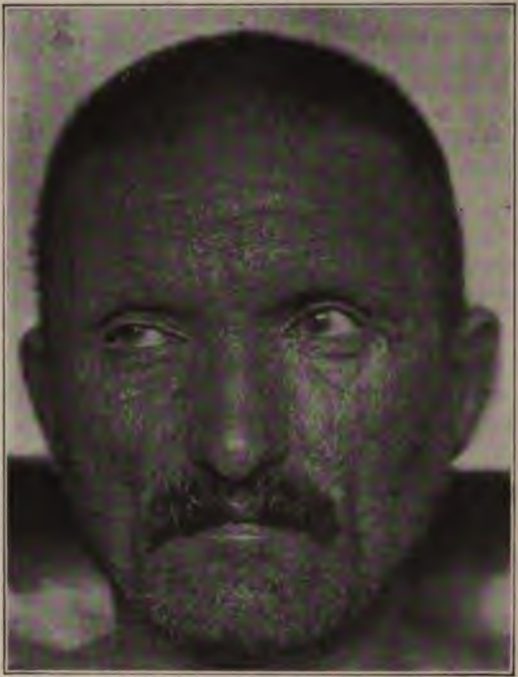

FIG. 70.—SUBACUTE ERYTHEMATOUS ECZEMA OF FACE. (Author's collection.)

In all forms ma, including t thematous, the re likely not to be form intensity o surface, but to be less mottled, or by innumerable p deeper color at t of the follicles.

Papular Ec Erythematous may pass into eczema by the ment over its su numerous infla papules. This, is not the usual of its develo Papular eczema ordinarily as dis flammatory papules or as groups of inflammatory papules distributed. The papules are small, pinhead-sized or larger, ac and from pink to red in color. As a result of scratching they to be excoriated, and even where not excoriated they become ra the rupture of minute acuminate vesicles which form upon th When grouped they form by their coalescence irregular patches elevated, indurated, and red, around the borders of which inflammatory lesions are found.

Vesicular Eczema.—Vesicular eczema, which is the typical the eruption, occurs as small acuminate vesicles on an inflam The vesicles may be discrete, but they frequently develop upon er tous patches in dense groups which coalesce. The vesicles in unless the process very quickly subsides, rupture, exposing the b mucous layer. According as the vesicles are discrete or conflue result discrete weeping points, or, as is usually the case, irregular red raw areas from which there exudes a clear gummy fluid which stif-

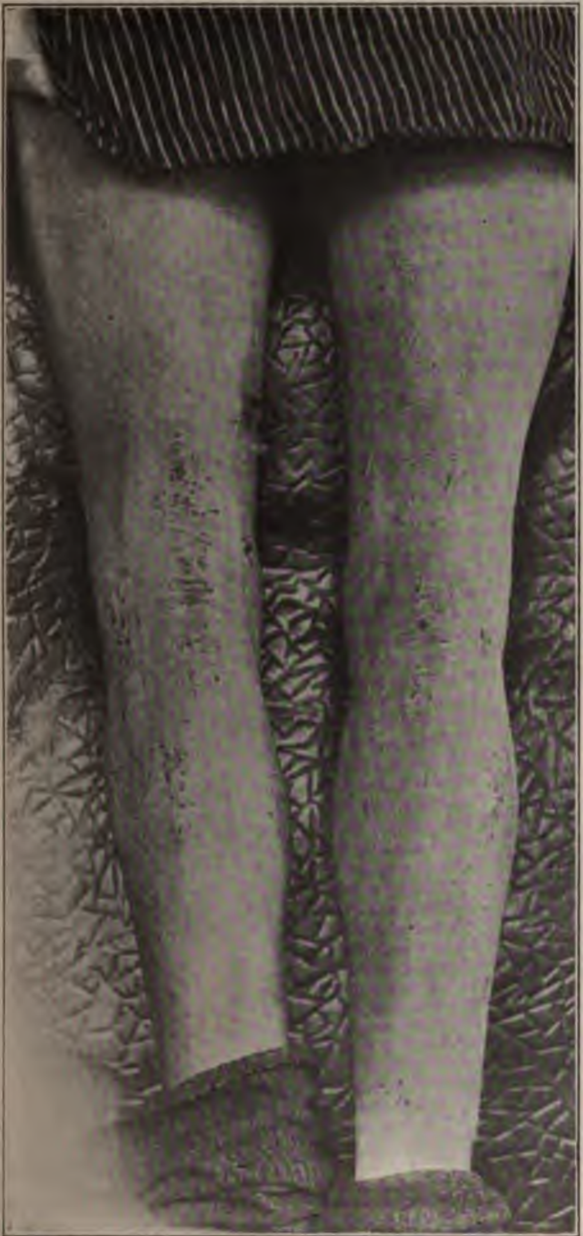

Fig. 71.—Chronic Papulo-vesicular Eczema of Legs in Poorly Developed Boy. (Author's collection.)

fens linen. Much has been made of this property of stiffenin which the excretion of eczema possesses. Blood serum contains ten per cent of albumin, and in addition the serum exuded in has its albumin in by the ruptured lial cells which tains. It is thi minous material stiffens upon dryi which makes the of eczema sticky, blood serum is sti will stiffen linen.

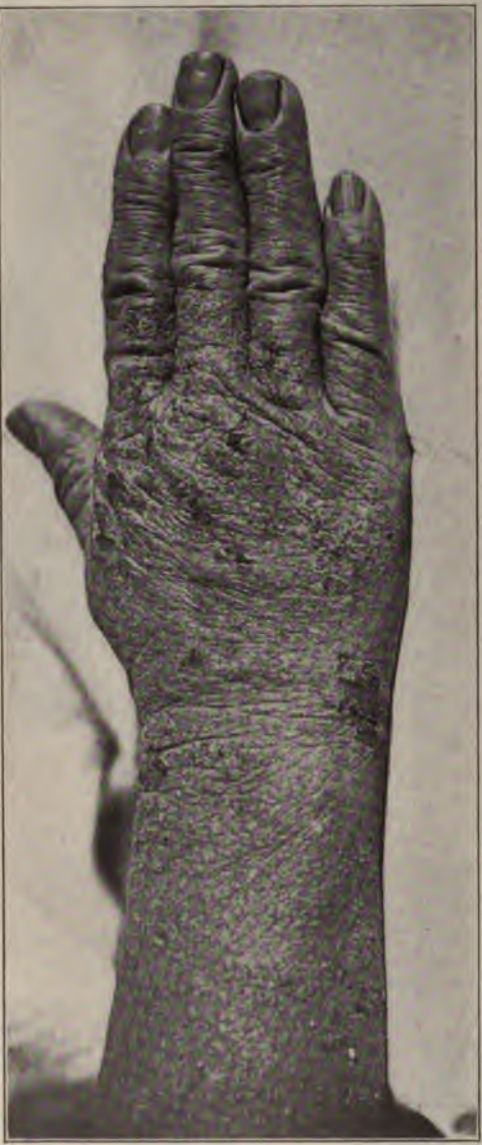
FIG. 72.—ACUTE VESICULAR ECZEMA OF HAND. (Schamberg's collection.)

The vesicula may be regarded acme of eczem the vesicles are grouped, as th likely to be, th weeping areas wh produced by the ture merge the into that of wee zema, *eczema rub* *eczema madidans* *madidus*, wet). a case we have i tion to the bright red, raw weeping a red border of zema ordinarily out gradually i healthy skin. border will be fou cles and some both of which gr become fewer tow outer edge. U weeping surface will be more or l ing of the secretion into yellowish, varnishlike crusts, usually wi admixture of pus as the result of infection. The process will intensity as the exciting factors vary and the picture thus will from time to time with no tendency to a definite course, its duration depending upon the duration of the exciting cause.

Pustular Eczema.—The contents of the vesicles in eczema may become purulent from the invasion of pus organisms. When this change occurs we have instead of vesicles small acuminate pustules on an inflammatory base. These rupture readily, leaving, as in vesicular eczema, raw red surfaces exuding a mixture of pus and serum. Pustular eczema frequently occurs as discrete pustules around the follicles, especially the hair follicles, where it shows as minute pustules pierced by hairs and surrounded by inflammatory areolæ. Where, as usual, the pustules are abundant, we have an inflammatory area dotted with small follicular pustules.

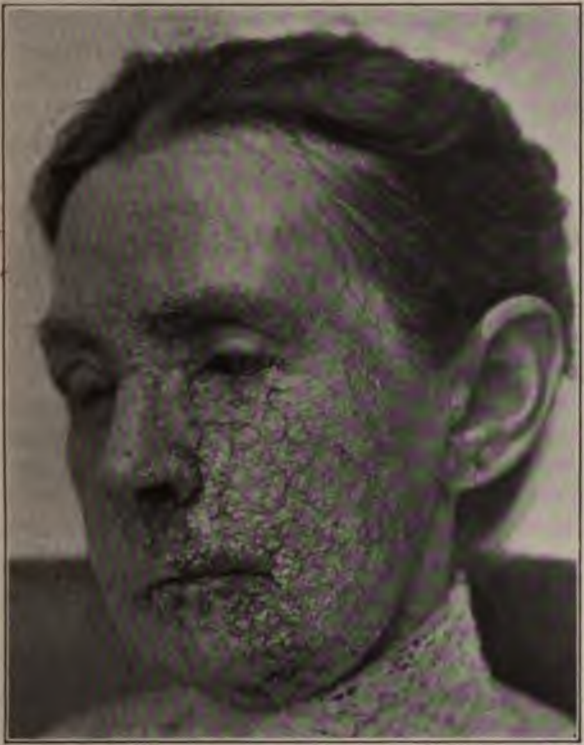

FIG. 73.—CRUSTED PUSTULAR ECZEMA. (Schamberg's collection.)

Subacute Eczemas.—All forms of eczema are apt to be persistent as the result of the continuation of the exciting cause. If the irritation remains acute the process will remain acute indefinitely. More frequently the process lapses into one of subacute intensity and the picture persists as a subacute dermatitis. In such cases we have persistent patches of erythematous dermatitis upon which there develop irregularly acute exacerbations of papules, vesicles, or pustules. In subacute persistent forms the formation of ill-developed epithelium is likely to be rapid and there is, as a result, increased exfoliation. If the process is of slight intensity the scaling may be rather abundant; this may be an exfoliation of small scales, but ordinarily it is exfoliation of large, rather thick, coarse scales. As a result of the persistence of the inflammatory process in these cases there is considerable infiltration of the skin. The picture on the whole is that of an ill-defined chronic dermatitis. The skin is faint pink to reddish in color, there is more or less exfoliation of horny scales, and the skin is thickened and indurated with its lines exaggerated. There may be no papular or vesicular lesions in these infiltrated patches, but with any exacerbation discrete papules and vesicles develop and excoriations will then be seen. This chronic, scaly, indurated eczema is a well-recognized picture in *eczema squamosum*.

The foregoing is but a bare outline sketch of the eruptions of eczema. To make the picture complete one must fill in the details of its extent, distribution, arrangement, and all the other features of the process which go to make up the symptom complex in any case. It is evident that the details of the disease may combine into an innumerable number of pictures, and yet in all of them it is easy to determine the essential elementary features.

Variations.—When eczema persists for a very long time in subacute form, the connective tissue and epithelial hyperplasia may become very marked. The skin may be greatly thickened and indurated as a result of the formation of new connective tissue, and in extreme cases the process may go on to conditions of well-marked elephantiasis, *eczema sclerosum*. In connection with this there may be hypertrophy of the papillæ and great increase in the epidermis, forming papillomatous, warty growths, *eczema verrucosum*, *eczema papillomatosum*. In such cases the skin may be unbroken, but there are apt to be fissures with a discharge, which from decomposition and admixture with decomposing horn cells is very offensive. These conditions are particularly apt to occur on the legs, where the process is favored by venous stasis. Where there is great thickening and induration of the tissues as the result of long-continued eczema on the legs, ulcers may form.

In thickened indurated patches of chronic eczema the skin is inelastic and of poor resistance, and as a consequence is apt to crack upon movement of the parts, forming fissures. The cracks upon the hands when the hands are chapped illustrate a moderate type of this condition. Where the induration is great upon parts subject to much motion, as the hands and feet, or about the deeper folds of the body, these fissures may extend entirely through the skin and be deep and painful, *eczema rimo-*

for the flexor surfaces, the muco-cutaneous junctures, and for the face and hands. In the case of the flexor surfaces and the muco-cutaneous junctures, the eruption most frequently occurs upon these sites because upon these surfaces the skin is thinner and more delicate and therefore responds to irritation more readily than does tougher skin. Around the muco-cutaneous junctures there is the added factor that the surfaces are exposed to the irritation of excretions. One factor in the frequency of the occurrence of eczema upon the hands and face is the fact that they are most exposed to external irritants. In the case of the face, however, and to a less extent the hands, we also have as a factor the fact that these parts are most sensitive to vasomotor disturbances of central origin, as is shown by the frequency with which vasomotor disturbances of all sorts occur upon them. Another site of predilection of eczema is the legs. Here the frequency of occurrence is explained by the venous congestion which renders the tissues particularly vulnerable.

Eczema has no definite course and no definite duration. Both depend upon the causes which produce the condition. An eczema may develop from a temporary exciting cause, pass through all the stages of eczema, and promptly subside, or it may subside from any of its stages. If it subsides, the erythema gradually disappears, there is some scaling of the surface, and a faint reddish stain is left which quickly disappears. But ordinarily it pursues no such short, definite course. It develops and continues with exacerbations and remissions, one day or one week better the next worse. This course is almost certain to persist until the excitin causes are removed, but even after these are apparently removed th process may still go on. For it is not only necessary to remove the caus but also to treat the dermatitis itself until the skin has had time to reac a condition in which it is able to withstand the ordinary external irritan to which it is constantly exposed.

The lesions of eczema appear irregularly or in crops without a regularity of sequence, with the result that one will frequently find one case lesions in all stages of their course. This occurrence at o time of lesions in various stages of evolution combined with the fact th the disease may show primarily at one time all sorts of inflammat lesions from papules to pustules accounts for the characteristic mul formity of the eruption.

When eczema continues for a considerable time the secondary infla matory changes which occur in the skin, rendering it thick and inelast may prove very persistent and in some cases, as in elephantiasis of legs, even be permanent. Ordinarily when eczema disappears it lea no trace. Ulceration of the corium does not occur, and therefore sc do not follow. On the legs it is frequent for eczema, as for all other flammatory processes there, to be followed by persistent pigmentati chiefly from extravasation of blood, and occasionally on the legs, when the process entirely subsides, instead of hypertrophy of the connective tissue there occurs atrophy, leaving the skin thin, atrophic, shiny, slightly red, and the constant site of recurrent eczema.

Complications.—The constant complication of eczema is infection with the common pus organisms, which changes its exudation from a serous to a sero-purulent or a purulent fluid. In such cases there is drying of dirty yellow, brownish, or greenish crusts upon the surface, with the development of superficial satellite pustules around the weeping areas. The amount of suppuration in such cases may be very abundant with the production of thick, heaped-up crusts. A not uncommon complication of these cases is boils, from infection of the hair follicles. It is common for the regional glands to be enlarged in extensive eczemas. If the eczema is pustular, infection of the glands not infrequently occurs with the formation of abscesses.

Eczema is constantly accompanied by itching. Where the process is of a very low grade of activity—as, for example, in chronic scaly eczema of the palms—it may be slight or even absent; but if the disease is at all extensive it is apt to be severe, and in some cases it amounts to torture that comes near being unbearable. It is the symptom for which the patient is insistent on relief. As a rule, it is most intense in those cases where there is not relief from tension by free exudation upon the surface, as in erythematous and papular eczema, and in vesicular lesions before rupture. The itching of pustular eczema and of fully developed weeping eczema is likely to be less. It is not continuous, but has waves of remission and exaggeration, and is usually worse at night. The scratching, which is irresistible, gives momentary relief, but results in injury to the tissues which increases the intensity and extent of the disease. In acute eczemas scratching may, unless controlled, serve to continue the disease indefinitely. In chronic infiltrated eczemas it is shown by the excoriations which even the most self-controlled patient will produce in his sleep unless the parts are so protected that they cannot be reached. The itching is exaggerated by all sorts of external irritation —by exposure to the air, by the contact of clothing, and particularly by contact with water. In some cases it taxes one's ingenuity to find any application that does not excite it. Occasionally there is substituted for the itching a feeling of tension, burning, or even pain, or these symptoms may be combined with itching.

In eczema the constitutional symptoms are remarkable for their absence. It is frequent for outbreaks of eczema to occur in patients who are run down and feeling out of sorts, but the disease itself causes practically no constitutional manifestations. A slight febrile reaction is occasionally observed, but it is by no means frequent. The patients are apt to be irritable and nervous and worn out from lack of sleep, and where the disease is extensive they are, as in other extensive inflammatory dermatoses, very sensitive to cold. There is no definite group of constitutional symptoms that characterize eczema.

Etiology.—Eczema constitutes approximately thirty to forty per cent of skin diseases. It occurs in both sexes, at all ages, and in all classes of society. From their greater exposure it is somewhat more frequent in males than in females. It is most frequent in infancy and during the active period of adult life. It is no respecter of persons, and the forms of it due to internal causes are probably quite as frequent in the well to do as in the poor; but on account of the conditions of occupation and of living of the poorer classes it is, on the whole, more frequent among those who do manual labor. The disease is not hereditary in the sense that it can be transmitted from parent to child, but there are certain conditions which predispose to eczema that are hereditary. The disease is more apt to occur in individuals with fair, thin skins than in those with dark, thick skins, for the reason that the thin skins are more sensitive. The skin may also be rendered more vulnerable and more subject to eczema from being congenitally defective, as in xeroderma and ichthyosis, in which conditions eczema is very prone to occur.

The causes of eczema are both constitutional and local. These supplement each other, and a hard-and-fast line cannot always be drawn between them. Either local or constitutional causes may, according to the intensity of their action, be predisposing or exciting.

External Causes.—The local causes are as numerous as the agents that can irritate the skin; they embrace all forms of irritants—chemical, mechanical, thermal, and, we must now add, actinic. Among the first class are numerous drugs and substances in common use which are chemically irritating. The drugs include the long list of substances which in various strengths cause an inflammatory reaction, such as mercury and its salts, sulphur, tar, chrysarobin, turpentine, mustard, dilute acids, and alkalies. Among substances in common use the first in frequency are perhaps soap and water, which render the skin less resistant by removing its fat and to a certain extent dissolving the horny epidermis. If used very frequently, therefore, especially if the weather is cold and if care is not taken to thoroughly dry the parts, they either excite dermatitis directly or put the skin in a condition in which a dermatitis is readily excited by other causes. The extreme examples of eczemas produced by soap and water we see in the occupation eczemas of washerwomen, barkeepers, and others whose vocations require that they have their hands continually wet. There are various other occupation eczemas, such as those arising from the handling of sugar, lime, turpentine, benzine, paints, and dyes. A familiar example is the eczema of the hands of surgeons which arises from the use of antiseptics. Nearly all of these occupation eczemas require as a factor in their production an unusual susceptibility, either a delicate skin or some departure from health which lowers the resistance, for these irritants are not ordinarily strong enough to excite inflammation in the normally resistant skin. Frequently-acting chemical irritants are decomposing secretions, such as decomposing sweat and sebaceous matter in the deep folds of the body, and the secretions from the various body orifices. A familiar illustration of eczema of this sort is seen around the genitals in children from the decomposition of urine. Another form of eczema due to urine is that seen in diabetes, where we have not only the effect of the irritating urine, but the lowered resistance of the tissues which occurs in the disease.

Eczemas due to chemical irritants are produced by numerous irritating plants, such as poison ivy, sumac, and primrose. These may present lesions indistinguishable from those of eczema, but they frequently produce a reaction so violent that it oversteps the bounds that we assign to eczema; such cases are considered under the various forms of dermatitis.

Among mechanical causes are friction and pressure, whose effect is most familiarly exemplified in the chafing produced by clothing or by the rubbing of adjacent parts together. These causes are comparatively trivial. The one very important mechanical cause of eczema is scratching. Dermatitis as the result of scratching is the familiar complication of all severely itching dermatoses, and it has to be taken into consideration wherever scratching is vigorous. It not only acts by producing excoriations and dermatitis from traumatism, but it also is likely to further complicate the process by infection with pus organisms. Scratching most frequently acts by increasing already existing eczemas or by superimposing an eczema upon another itching dermatosis, or it may be the most important if not the sole exciting factor of eczema in an itching dermatosis, as, for example, in scabies.

As will be seen, the rôle of bacteria as a primary cause of eczema is probably not important, but in all weeping eczemas we have to take into consideration the secondary effect which is produced by infection with pus organisms. There is no reason to believe that eczema is the result of a definite organism or that it is ever contagious. It may be associated with diseases, like scabies, which are contagious and, when they are transmitted, may occur secondarily. The action of the grosser parasites which prey upon the skin and cause severe itching is important in producing eczema. In these cases, however, the eczema is a sequel due solely to scratching, and these conditions are properly considered under parasitic diseases.

Sunburn, the dermatitis excited by artificial heat, and x-ray dermatitis are considered separately.

Internal Causes.—The internal causes of eczema are perhaps more frequently predisposing than exciting, acting by causing a lowered resistance which involves the skin, as well as other tissues, and renders it less able to withstand the ordinary insults to which it is subject. Internal causes, however, are undoubtedly in some cases of sufficient intensity alone to excite eczema. The eczemas so excited are vasomotor disturbances quite analogous to such vasomotor disturbances as erythema multiforme. Some of these forms of eczema are, in my opinion, sufficiently distinctive in their clinical characteristics to separate them as definite clinical entities. (Cf. Eczema of the Hands and Face, and Infantile Eczema.)

In the vast majority of instances the internal causes of eczema are toxic or metabolic. In a certain proportion they are distinctly of nervous character.

It is a frequent observation that patients with eczema are not in a vigorous condition of health. There may be no tangible pathological condition other than the eczema, but the patients are run down, overworked, or depressed mentally or physically. Of the definite disturbances of health, disturbances of digestion with the consequent autointoxication from the intestinal tract are most frequently active. All of the intestinal disturbances which are associated with putrefactive changes may act in this way. The most frequent condition, however, is that of intestinal atony with the consequent constipation and intestinal fermentation. A potent factor in exciting attacks in such cases is habitual overeating with the whole train of digestive disturbances which follows it. The various metabolic disturbances, which we include under lithæmia and the gouty and rheumatic diatheses and which are all essentially toxæmias resulting from a disturbance of the balance between excretion and assimilation, perhaps act in some cases as causes of eczema independently of the intestinal complications which usually accompany them. But they are so bound up with digestive disturbances that it is difficult to determine the extent of their influence. These conditions, like all other metabolic disturbances, require correction as far as possible in the treatment of the eczema. The association of eczema with defective urinary elimination is not as definite as would be expected. The association of nephritis with eczema is occasionally observed. Glycosuria with eczema is of more frequent occurrence. According to Crocker and Duhring, this association is rare, but in my experience the connection has been very definite in a certain number of cases of intolerable eczema about the genitals, in which there seemed every reason to believe that the irritation was not the result of contact of the urine with the parts. Eczema is not infrequently associated with rickets and similar nutritional disturbances. In these cases the eczema apparently results partly from gastrointestinal disturbances and partly from the lowered resistance of the skin, which is only one manifestation of the general condition of lowered resistance of these patients. Under the name of *eczema rubrum scrofulosorum*[1] Crocker has called especial attention to a weeping, indolent, purulent eczema which occurs upon the feet and the lower part of the legs in markedly scrofulous patients. The lesions occur in sharply defined patches, and apparently are the result of infection of skin of low resistance.

The relationship of the nervous system to eczema is even less tangible than that of nutritional and toxic disturbances, but that it is an important factor in the etiology of eczema is impressed upon the notice of everyone who has observed many cases of eczema. As Hebra long since, and Bulkley and others more recently, have pointed out, "faulty innervation" is an important factor in many cases. It is a cause that cannot easily be proved, and it is apt to be overworked as an explanation, but it is none the less of real importance. The patients in such cases are overwrought, depressed mentally or physically, neurasthenic, and present all sorts of evidences of unstable nervous equilibrium. In some cases the beginning can be traced definitely to emotional disturbances, as severe mental shock. Rarely it is associated with hysteria, and in a few cases the eruption has closely followed nerve distribution. It is apparently, in some cases, the result of reflex irritation. The genito-urinary tract has frequently been compelled to bear the responsibility for the occurrence of eczema, and in rare cases the connection, especially with uterine disturbances, seems definite, as in cases associated with pregnancy, lactation, uterine tumors, and the climacteric. Reflex nervous eczemas from irritation arising in other organs apparently sometimes occur. Abramitcheff, for example, has recorded a case of persistent eczema which was relieved upon wearing a suitable belt to support a movable kidney, and which recurred whenever the support was withdrawn. Reflex irritation from dentition has frequently been invoked to explain infantile eczema. There seems every reason to believe that the cause of this eczema is not reflex irritation, but autointoxication from the digestive tract.

Bulkley and Taylor have emphasized the connection of eczema with asthma, and a good many times I have seen persistent forms of symmetrical eczema in asthmatic subjects, where there seemed good ground for believing that the eczema was a manifestation of the same nervous disturbance that caused the asthma.

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[1] Crocker, p. 192.—Sanglé, "Étude sur l'Eczéma scrofuleux," *Thèse de Paris*, 1880.

Finally, there occasionally come persistent, rebellious cases of eczema which baffle all attempts at discovery of their cause; cases whose course the most expert completely fail to influence. In one such case of eczema of the face, which at one time was under my care, the disease disappeared subsequently upon the removal of normal ovaries, and most of such cases, in my opinion, depend upon some deep-seated disturbance of innervation.

Speaking broadly, I believe that all of the internal causes which have been referred to under the etiology of urticaria and erythema multiforme may act equally in producing eczemas; and in the same way by causing vasomotor disturbances. The association of eczema and urticaria, aside from the secondary eczema produced by scratching, is not uncommon, and I have seen typical papular erythema multiforme on the back of the hands alternate with eczema of the same parts.

According to Duhring, eczema usually disappears during an attack of a febrile disease, like typhoid fever, to reappear after the acute illness is past. The same disappearance of eczema upon vaccination is sometimes observed. The opposite effect of acute disease and of many chronic diseases is often seen, eczema developing in patients who are reduced from a recent acute illness or from some chronic disease.

The question has often been raised of the danger of suppressing an eczema and thereby causing a metastasis of the inflammatory process to some other part.[1] Cases where there seemed to have been a metastasis of this sort have been recorded by Brooke, Brocq, Gaucher, Duhring, and others. If such metastasis ever occurs it must be of extraordinary rarity. In my opinion the danger is hypothetical, and if it does exist is so extremely remote that it may be absolutely neglected.

Pathology.—The reasons for believing that eczema is not a disease of specific etiology have already been considered. Where eczema is of internal origin it is a vasomotor disturbance, probably in most cases of central, but perhaps occasionally of peripheral origin. The parts played by autointoxications, metabolic disturbances, and various disturbances in causing the vasomotor disturbances which produce eczema, have been considered under etiology. The rôle of bacteria in the causation of eczema has already been referred to. For many years Unna was insistent that his morococcus was the specific organism of the disease. At the International Congress of Dermatology in Paris, in 1900, in a discussion upon the etiology of eczema Unna was practically compelled to abandon all of his contentions in favor of the morococcus. As Sabouraud has shown, the morococcus belongs to the staphylococcus group, and there seems to be practically no doubt that it is the staphylococcus epidermidis albus of Welch, which in turn is not clearly distinguished from the ordinary staphylococcus albus. The fluid of unruptured vesicles of eczema is sterile, and bacteria are only associated with the later evolution of the lesions.

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[1] Butte, *Ann. de Ther. Derm. et Syph.*, vol. iv, No. 7; *Monat.* XXXIX, p. 163.—Feer, *Corr. Bl. f. schweiz. Ärzte*, 1904, No. 1; *Monat.* XXXIX, p. 420.

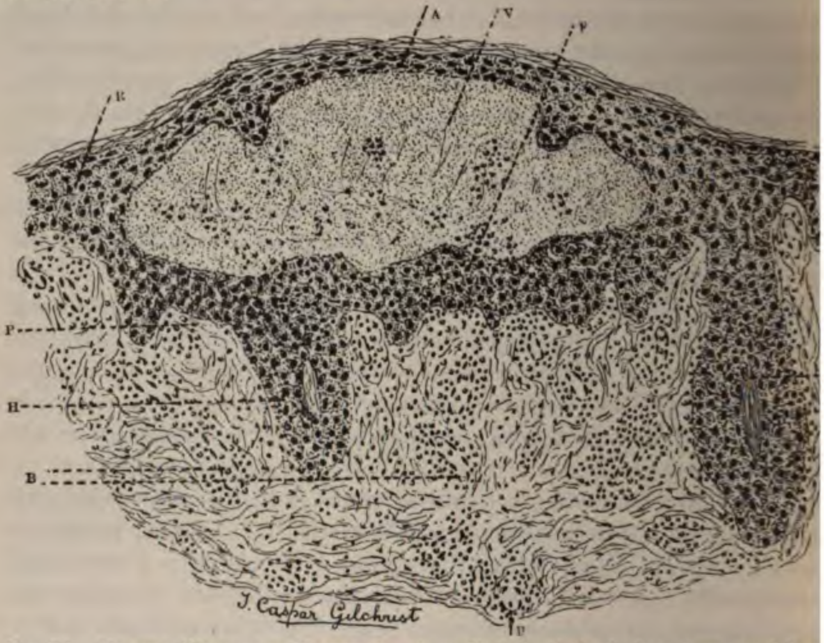

Fig. 76.—Eczema (Vesicular Variety). A small pinhead-sized, clear, tense vesicle w excised from the tip of the right ear. The vesicle was surrounded by a slight area inflammation. In the same patient there were other distinct evidences of ecze vesiculosum et pustulosum, with crusts around the ears and neck. The vesicle (is seen to be situated in the middle of the stratum Mucosum (*R*), and took its orig therefore in this layer. The contents of the vesicle consisted of numerous pol nuclear leucocytes (horseshoe-shaped in the drawing), a few detached epithelial cel some strands of fibrin, and a large quantity of coagulated albumen (serous exud tion); very few round, mononuclear cells were seen in this region. The epiderm cells forming the roof (*A*) of the vesicle were swollen and were elongated; the int epithelial spaces were also much wider than normal. Numerous polynuclear leuc cytes had invaded the epidermal floor (*F*) of the vesicle, and particularly the epith lium of the hair follicles (*H*), which were also much swollen. The corium, especial the upper portion, showed marked evidences of acute inflammation. The papil (*P*) were swollen and infiltrated with polynuclear leucocytes. The blood vessels (were all markedly dilated, and contained and were surrounded by numerous pol nuclear cells. Mononuclear (lymphoid) cells were also numerous, especially in t neighborhood of the vessels. One hair follicle (*H*) was implicated in this vesicul formation, but the sweat ducts were unaffected. Magnified about 100 diamete (Dr. T. Caspar Gilchrist's case, section, and description.) (From Duhring.)

The anatomy of eczema has been investigated by many observer Among them may be mentioned Hebra, Kaposi, Biesiadecki, Robinso Unna, and Gilchrist. There has been strong contention, especially those who would make of eczema a specific disease, that the prima

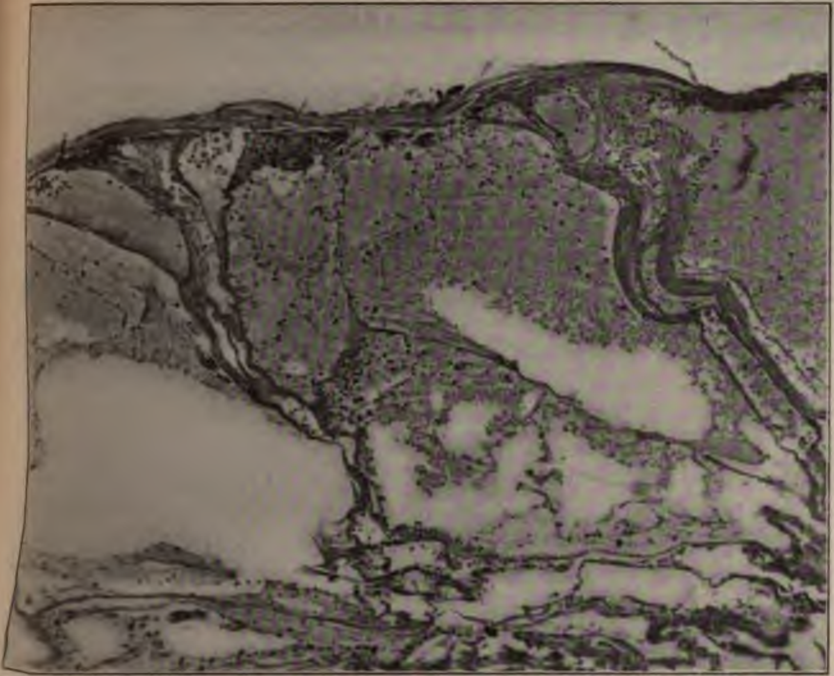

FIG. 1.—ECZEMA VESICLE. Multilocular vesicle beneath horny layer of epidermis. Roof consists of horny epidermis from which strands of epithelial cells connect with floor of vesicle. Contents of vesicle consist of granular material and a few polymorphonuclear leucocytes. × 30. (Author's collection.)

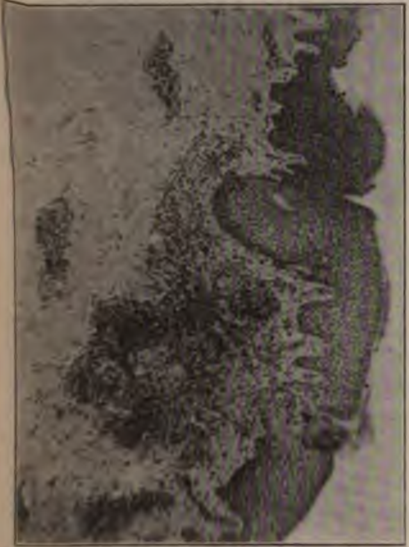

FIG. 2.—ECZEMA PAPULE. Showing a minute area of inflammatory infiltration in corium. × 60. (Author's collection.)

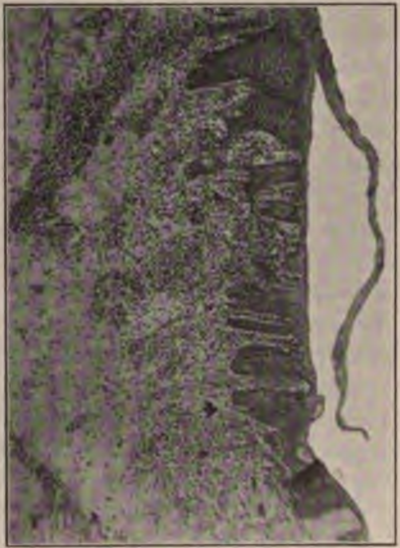

FIG. 3.—SCALING ECZEMA. Inflammatory infiltration of corium with scaling of epidermis. × 60. (Author's collection.)

HISTOLOGY OF ECZEMA

changes in the process are in the epidermis and that the changes in the papillary layer are secondary. All of the changes of eczema are those of an inflammatory process. Even the most enthusiastic advocates cannot pretend to produce a section of an eczema lesion not showing inflammatory changes in the corium, and a consideration of such sections as are here reproduced and of the ordinary course of an inflammatory process leaves no room for doubt that the changes are chiefly, as Crocker says, "in acute eczema primarily in the papillary layer, afterwards in the epidermis, and, if of sufficient duration, the deep portion of the corium may be involved."

In acute eczema the corium shows all the changes of an acute inflammatory process. The epidermis is œdematous and invaded by polymorpho- and mononuclear leucocytes, the cells are swollen, more or less degenerated, and, from the pressure of the œdema, transversely elongated. The vesicles are formed by rupture of the epidermal cells and intercellular processes and contain serum, leucocytes, fragments of epidermal cells, coagulated albumin, and strands of fibrin. If the condition persists, proliferation of the rete is more rapid than normal, mitotic figures are numerous, and marked hypertrophy of the rete may occur (acanthosis). At the same time the cells do not undergo perfect cornification (parakeratosis), but remain softer than normal and are exfoliated freely.

In acute eczema of the ordinary type the process is confined to the upper part of the corium, and in all forms usually remains most marked in the papillary layer. It usually begins around the follicles and remains most intense there. When the disease persists for a considerable time the process invades the entire corium. In eczemas of subacute intensity acanthosis and parakeratosis are most marked, and the changes in the horny layer are those of an inflammatory process of less activity. Where the disease persists of subacute intensity secondary sclerosis of the corium occurs. The changes in the epidermis are such as have already been described, but more marked. The line of demarcation between the epidermis and corium may become vague from the disintegration of the lower cells of the rete. The papillæ are usually hypertrophied, although rarely they may be obliterated. The blood vessels and lymphatics are dilated, but in the cases where the greatest proliferation of connective tissue takes place obstruction of the lymphatics is usually the cause. Where the disease is long continued these changes may extend well down to the subcutaneous layer. The histological pictures which the various forms of eczema show are well illustrated in the accompanying reproductions of sections and descriptions from Duhring.

Diagnosis.—While eczema appears in most varied clinical pictures in different cases, it nevertheless presents a complex which usually sharply differentiates it from other dermatoses. It must be remembered, however, that the mere determination that an eruption is eczema does not carry one very far. It at least excludes all specific dermatoses, but it does not mean more than the establishment of the fact that the eruption is a simple dermatitis. After this is established there is left for solution the frequently much more complex problem of its etiology.

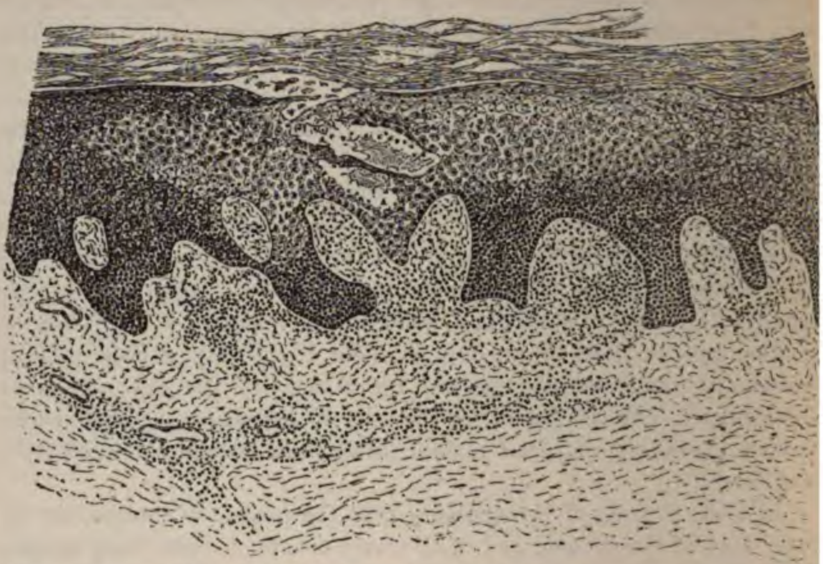

FIG. 77.—ECZEMA (VESICULAR VARIETY, NEURITIC TYPE.) Section from palmar surfa of a finger. The corneous layer shows lacunæ, due to the separation in places the strata. This is not necessarily a part of the process of eczema at this stage, b is usually present in later stages. The stratum lucidum is ruptured at one pla above the vesicle *a*, and a space containing serum and a few leucocytes is seen; represents the earliest stage at which a vesicle is observable. The vesicle lies in t mucous layer. The contents consist of serum and leucocytes. In this section t coagulated albumen is well shown. The vesicle forms by transudation from t blood vessels into the mucous layer at a given point and pushes the cells apart, co pressing them and causing them to elongate. In this drawing the mucous lay beneath the vesicle shows signs of œdema, and this occurs in a considerable ar around the vesicle. The cell body undergoes molecular changes and does not sta well. The lighter part of the mucous layer in the drawing shows the extent of t molecular change and corresponds to the area of inflammation in the papillary lay of the corium. The papillæ show marked serous transudation, dilated blood vess and invasion of leucocytes. The limit of the process is tolerably well defined, bo in the corium and in the epidermis. (Dr. A. R. Robinson's section, drawing, a description.) (From Duhring.)

The characteristic features of eczema are the evidences of a simp inflammatory process, the marked tendency to itching, the multiformit of the lesions, the indefinite course, and the predilection of the eru tion for parts where the skin is tender or especially exposed to irritant The eruption shows all the characteristics of an inflammatory proces

The lesions are such as can be produced by external irritants; they show the redness of hyperæmia; there is more or less swelling from the inflammatory exudate; and there is either scaling, or destruction of the horny layer with the appearance of weeping surfaces upon which crusts tend to form. The tendency to itching is marked, and in all except the most indolent cases is apt to be intense. The eruption appears with no regularity and runs an indefinite course; weeping surfaces may persist with no tendency to self-limitation. As a result of this indefinite course the eruption is multiform. It shows all sorts of inflammatory lesions, and in persistent cases also the sequelæ of inflammation in the skin. The multiformity of the eruption is one of the striking characteristics of eczema. Syphilis is the only definite disease which presents the same multiformity of lesions. There is a predilection for surfaces where the skin is delicate (the flexures and inner surfaces), for the muco-cutaneous junctures, and for exposed parts (the hands and face). This feature, while fairly constant, is not an essential feature of the disease and may be lacking, for, of course, dermatitis may occur in the skin of any part.

From the variety of its appearances eczema presents in different cases a superficial similarity to almost every inflammatory dermatosis; a similarity, however, which is usually not confusing if a careful search is made for its characteristic feature. The mistake most frequently made is not in failing to recognize an eczema, but in failing in mixed cases to recognize that the eczema is a secondary condition engrafted upon some other itching dermatosis.

There is a possibility of confusing eczema with erythema multiforme, urticaria, prurigo, the various forms of herpes, dermatitis herpetiformis, pemphigus, impetigo contagiosa, erysipelas, rosacea, psoriasis, seborrhœic eczema, lichen planus, pityriasis rubra, exfoliative dermatitis, lupus erythematosus, scabies, pediculosis, favus, ringworm, miliaria, syphilis, and occasionally with other inflammatory dermatoses. The list is apparently formidable, but as a matter of fact it is not often that the resemblance between an uncomplicated eczema and any of these affections is close enough to give any difficulty in differentiation, if the characteristic features of the different diseases are borne in mind. For the characteristic features of these various diseases it is best to refer to their descriptions, although some of them are briefly summarized below.

Erythema multiforme in its various forms runs a definite course, has characteristic and peculiar lesions, itches slightly or not at all, and has a characteristic distribution.

Urticaria is characterized by wheals which are evanescent, and can usually be produced artificially. It may be accompanied by a secondary dermatitis due to scratching.

In prurigo there is secondary dermatitis. In addition there are the history of continuance since early childhood, the peculiar distribution on the legs, and the presence of prurigo papules.

The characteristics of herpes and herpes zoster are so sharp that there should never be any difficulty in a differential diagnosis from eczema.

In dermatitis herpetiformis the lesions appear in herpetiform groups with a tendency to ring formation, the individual lesions show no tendency to rupture, and go through a regular course, bullæ are common, and the vesicles which occur are not the pinhead size, acuminate vesicles seen in eczema, and do not rupture.

In pemphigus there is always the occurrence of spontaneous bullæ, which are evident either by their actual presence or by their traces, shown by fringes of undermined epidermis.

The differential diagnosis from the several other diseases which have not yet been considered will be taken up with them.

Prognosis.—The natural tendency of eczema is to pursue an indefinite, irregular course. The cases tend to continue until the exciting causes are removed, and even then may persist unless suitably treated. The result of treatment depends very largely upon the ability to remove the underlying causes. Where these are apparent and easily overcome the disease yields readily to treatment. Where they are difficult or impossible of removal, or where they are indeterminate, results are very much less certain. Even in many such cases, however, judicious symptomatic treatment is successful. But some rare cases of eczema, dependent upon irremediable or undiscoverable internal causes, prove absolutely rebellious to treatment, and either persist through life or gradually wear themselves out. In such cases the best that can be done is to give partial relief from the symptoms, and it is practically never impossible to do at least thus much.

Treatment. **Internal Treatment.**—The internal treatment of eczema is almost entirely symptomatic, and consists in the rational treatment of the various disturbances of general health which have been considered under etiology. In all cases it is desirable to correct as far as possible any disturbances of the general health which may exist, although, truth to tell, we are fortunately able chiefly by local treatment to cure many cases of eczema in which we find constitutional disturbances that we cannot remedy. It is not intended by this to belittle the importance of constitutional treatment of eczema. The relief of internal conditions is in many cases a prerequisite to successful management of the disease, and in some cases in which this is impossible the eczema proves beyond cure.

In the great majority of eczemas of internal origin the constitutional conditions which require regulation are toxæmias, in most cases due to digestive disturbances, in some cases of other forms. It goes without saying that the internal treatment of such conditions is a distinct problem in each case. In a majority of cases of eczema a careful regulation of the diet is desirable. In patients who are in a lowered condition of health a liberal diet with all the tonic adjuvants may be indicated, but as a rule in eczemas of internal origin one of the most useful things that we have to do is to curb habits of so-called good living; and the most potent thing in this connection often is to materially reduce the quantity of food that the patient is taking. After this the next thing is to regulate the quality of the diet as carefully as practicable. In intestinal dyspepsia particular attention has to be given to limiting the consumption of starchy foods and vegetables which produce intestinal fermentation. The excessive consumption of sugar is a matter that is especially to be looked out for, and in some cases it is almost solely responsible for the continuance of subacute eczemas. Among articles the use of which it is necessary to restrict are pastries and cakes, hot breads (especially that American iniquity, hot griddle cakes and sirup), sweets and confections, salt meats, fried stuffs, cooked-over meats (hashes and made dishes), cheese, and highly seasoned foods generally. In acute eczemas occasionally much can be done by placing the patient on a light hospital diet, in some cases even on a liquid or milk diet, and in such cases hot liquids should be avoided. The use of coffee and tea should be restricted. The immoderate use of tea is, I think, particularly harmful in certain toxic cases. In certain cases in which there is a nervous factor in the causation, smoking may bring on an attack or cause the disease to persist, and has to be interdicted or allowed only in the greatest moderation. As a rule, alcohol is better entirely avoided unless the patient is old or weak and accustomed to taking it, or unless he is thoroughly habituated to it and would suffer a severe shock upon its entire withdrawal. If alcohol must be taken, a light, dry wine or spirits largely diluted are least objectionable. In debilitated patients beer may occasionally be a good stomachic tonic, but in toxic cases it is objectionable, and champagne extremely so.

To increase elimination and as an aid in overcoming constipation the abundant drinking of water is very useful. For the relief of constipation a large draught of water (preferably hot, with a little lemon juice or salt to give it taste) before breakfast is a very useful measure, and ordinarily the patient should be instructed to drink the conventional two quarts of water or more daily. Contrary to the ordinary opinion, I do not believe it is harmful if the water is drunk freely during meals, except in so far as it stimulates the appetite.

Attention should be paid in chronic cases to the discovery and correction as far as possible of improper habits of living. Exercise, lif in the sunlight and fresh air, diversions and relief from care, perhap entire relief from business, and change of scene are all possible factor of treatment to be considered. For the relief of constipation calistheni exercises of the abdominal muscles and abdominal massage are frequentl serviceable. A good way for the patient to give himself abdominal massage is to get a large tenpin ball, but one not heavy enough to caus discomfort, and to roll it around the abdomen in the direction of th colon for five to ten minutes daily. In acute eczemas means should b taken as far as possible to afford sleep, rest, and relief from itching In extensive acute cases patients do best in bed and under the genera regulations that apply to acute illnesses. Sedatives have to be used wit caution, but in some cases benefit is gotten from the intelligent use o asafœtida, valerian, the bromides, chloral, sulphonal, trional, atropin hyoscyamus, and other sedatives. Opium does not give us the service i these cases that it does in painful conditions, and ordinarily has to b avoided.

In all eczemas it is a good rule at the beginning of treatment t clean out the bowels thoroughly and afterwards to keep them well ope For the former purpose mercurial cathartics are most satisfactory; fo adults calomel or blue mass, for infants calomel or gray powder. I all toxic cases the most useful cathartics are the mercurials, and benef is likely to be gained from their repetition every few days during tl existence of the eczema. A favorite form is calomel in doses of $\frac{1}{10}$ $\frac{1}{4}$ grain combined with 1 or 2 grains of bicarbonate of soda. This given at intervals of half an hour to two hours until one or two grai are taken or until catharsis takes place. After the mercurials in us fulness come the saline aperients, and here the natural mineral wate often furnish an eligible form of administration. In chronic cons pation and in lithæmic patients I have found, following the suggesti of Sir Henry Thompson, that the natural Carlsbad water is a partic larly useful aperient water. Most of the bottled natural aperient wate both the American and imported, are satisfactory, but offer no essent advantages over the pure salts when they are taken well diluted. Amo the saline aperients, sodium phosphate, especially in the effervescent m tures, is very useful. Eligible prescriptions are the following:

℞ Sodii phosphatis	℥i;
Acidi phosphorici dil	fℨv;
Syr. zingiberis	f℥iss;
Aq. menth. piperit. . . . ad	f℥vj.

M. Sig.: A tablespoonful, in a large glass of water, t.i.d.

℞ Magnesii sulphatis	℥i–iss;
Ferri sulphatis	gr. iv;
Aq. menth. piperit ad	f℥iv.

M. Sig.: A tablespoonful, half an hour before breakfast, or a teaspoonful t.i.d., before or after meals, well diluted.

℞ Magnesii sulphatis	℥i;
Sodii chloridi	ʒi;
Ferri sulphatis	gr. iv;
Tinct. cardamomi comp.	f℥i;
Aq. dest. ad	f℥iv.

M. Sig.: A tablespoonful in a large glass of water before breakfast. (Duhring.)

These two latter are very useful mixtures for many conditions in which we want to give iron to constipated patients.

A mixture recommended by Stelwagon where salines are not well taken is as follows:

℞ Sodii salicylat.	ʒi–ij;
Ext. cascaræ sagradæ fl.	fʒi–iv;
Tinct. nucis vomicæ	fʒij–iv;
Tinct. cardamom. or tinct. gentian q. s. ad	fʒiij.

M. Sig.: A teaspoonful in water after each meal.

Other aperients which are called into service as alteratives are compound licorice powder, sirup of senna, infusion of senna or, better, senna tea made at home, fluid extract of cascara sagrada, podophyllin, colocynth, and the aloin, belladonna, and strychnine pill.

In the eczemas of gouty individuals, alkalies and saline aperients and colchicum give good service.

In cases which are dependent upon vasomotor disturbances, Crocker recommends counter irritation over the vasomotor centers of the involved areas by means of dry heat, mustard plaster, or liquor epispasticus. Stelwagon recommends galvanism up and down the spine, and Hyde the application of the brush discharge from a static machine. A high-frequency current may also be used for the same purpose. My experience with these methods has not been favorable. In rebellious eczemas of vasomotor origin, both of chronic and acute intensity, great benefit may be derived from careful, light exposures of the involved parts to x-rays. These exposures should be given very cautiously and kept well short of producing any visible effect upon the skin. If so used, they frequently prove a veritable boon in otherwise intractable cases. The effect upon itching is apt to be prompt and persistent, and in many cases the eruption fades out rapidly under the treatment. In a few rebellious cases, especially of chronic eczema of the face, I have seen this measure prove perfectly useless, but I have never seen it make the condition worse. It should not be used, however, unless the operator is perfectly sure that he can use it without overdoing.

The field of usefulness of specific medication is unfortunately very limited. Pilocarpin, preferably given hypodermically, in doses sufficient to set up slight perspiration, $\frac{1}{16}$ to $\frac{1}{8}$ grain, is sometimes useful in relieving itching and sometimes, I believe, positively benefits the condition. It is chiefly used in extensive erythematous and papular eczemas in patients with dry skins. In acute eczema Hutchinson and Morris highly recommend wine of antimony, about 5 drops, t.i.d., and I believe it is sometimes of value. Crocker uses oil of turpentine where there is no irritation of the alimentary tract or urinary organs. It is given in an emulsion of mucilage of acacia in doses at first of not more than 10 minims, t.i.d., p.c., the last dose not later than 6 P.M. With it barley water or other diluents should be drunk freely—"not less than a quart a day." The dose may be increased up to 20 or 30 drops in some cases without producing disturbing symptoms. Various other remedies have the recommendation of good authorities: fluid extract of viola tricolor, 10 to 15 drops in adults and 1 to 5 drops in children, once or twice daily (Piffard); tar, 10 to 30 drops, t.i.d. (Duhring, McCall Anderson); ergotin (Lewin, Jarisch); in angioneurotic cases; ichthyol, 5 to 10 grains, t.i.d. (Unna). But these specifics are apt to be useless.

In connection with the use of specific remedies in eczema it is necessary to call attention to the fact that arsenic is not generally indicated. Arsenic is for all practical purposes a stimulant to the skin, and is contraindicated in acute eczemas, just as local stimulants are contraindicated. It does not seem to be useful even in chronic squamous eczemas, where, from analogy with other conditions in which it is useful, benefit might be expected from it. If used at all in eczema it should be used on account of general indications for it as a tonic, and even then it should be given with caution.

Local Treatment.—The local treatment of eczema embraces almost all the means which are used in treating inflammatory dermatoses and in its entire scope covers a large part of the local therapeutics of skin diseases.

In the local treatment of eczema we have two broad purposes in view: in acute eczemas the purpose is to get rid of irritation and subdue inflammation; in chronic eczemas, also, the attainment of these ends may be our aim, but frequently we have the added problem of getting rid of changes produced by a chronic inflammatory process. For these we are compelled to avail ourselves cautiously of stimulation and the exciting of a greater or less degree of acute inflammatory reaction. In addition to these essential indications in the treatment of eczema, we often have to direct measures especially to the relief of itching.

In the soothing treatment of acute eczemas the first indication is to remove as far as possible all external sources of irritation. To this end measures have to be taken to protect the surface from the air, from water, and from other external irritants, and to keep it clean. Protection from the air is furnished by all of the various sorts of local applications which are used. As regards the use of water in eczema, acutely inflamed areas should not be washed at all with water, and certainly not with water and soap. They can be cleaned satisfactorily with liquid vaseline or olive oil. A more agreeable but also somewhat more irritating application for cleansing is a tragacanth cream—tragacanth (one per cent) and glycerine (three per cent) and borax (two per cent), in water. Where general baths are necessary in eczema, mucilaginous baths are least irritating, such as are made with starch, linseed meal, oatmeal, bran, gelatine, or marshmallow, 1 or 2 pounds to the bath. Next to these are alkaline baths, soda bicarbonate (6 to 12 ounces), or borax (2 to 6 ounces to the bath). But in generalized acute eczemas it is better to avoid general bathing and to be content until the process somewhat subsides with cleaning the surface with oils or with tragacanth cream. It may be remarked here that while the occasional application of water, especially when combined with soap, is irritating to eczemas, the use of medicated lotions may be soothing if they are continuously applied in the form of wet dressings, or if their application is immediately followed by ointments or similar protective fatty dressings.

In the beginning of treatment of a suppurating eczema upon which there accumulate pus crusts and dirt, the first indication is to get rid of these. For this purpose we may apply a dressing of olive oil or of vaseline to which may be added four to six per cent of boric acid, or one half per cent of carbolic acid. Another equally satisfactory way is to apply wet dressings of boric acid or sodium bicarbonate, about 2 drams to the pint. Or this may be done, but no more satisfactorily, by the use of a boric-acid starch poultice. After the surface is cleansed of the accumulated crusts there is left a weeping surface which is still suppurating. Until the suppuration is relieved wet dressings furnish a good method of treatment, and for this purpose dressings of boric acid (four per cent), ichthyol (two to five per cent), or of normal salt solution are as good as any. Another plan of treatment of pustular eczemas is by the use of ointments containing antiseptics, say, for example, boric acid, 60 grains to the ounce; iodoform, 3 to 5 grains; aristol, 20 to 40 grains; sulphur, 5 to 20 grains; ichthyol, 10 to 60 grains; or better than any of these usually, ammoniated mercury or yellow oxide of mercury, 10 to 20 grains to the ounce. Another good plan is to spray the surface by means of an atomizer with a solution of peroxide of hydrogen, either full strength or diluted with an equal quantity of water, after which the surface is wiped clean and covered with diachylon ointment.

With the relief of the suppuration the situation which confronts us is that of a weeping or vesicular eczema. We assume here, for consecutiveness of description, that this is a sequel of a pustular eczema, but as a matter of fact it may as well be, and is more frequently, the primary condition which we are called upon to treat. In weeping or vesicular eczema the indications for allaying irritation, and especially for relieving itching, are usually more pressing, and treatment therefore must be as soothing as possible. According to convenience or efficiency some cases are treated with lotions, some with fatty applications, and some with combinations of the two.

For wet dressings, among the most soothing applications are boric-acid solution, solution of aluminum acetate (liquor Burrowii), and lead and opium wash in the strength of tincture of opium and solution of subacetate of lead each 10 to 30 drops to the ounce of water or, better, in fresh milk. These preparations may advantageously be applied continuously as wet dressings. Lotions, however, are frequently most useful when dabbed upon the surface or applied for a short time, and then supplemented by the application of a soothing ointment. Among soothing applications which are antipruritic and, most of them, slightly astringent are the distilled extract of witch-hazel, fluid extract of grindelia robusta, 5 to 10 minims (Duhring) to the ounce; ichthyol, 5 to 30 minims; carbolic acid, 5 to 15 grains; creosote, 5 to 15 grains; thymol, 5 to 15 grains; creolin, 5 to 15 minims; camphor chloral, 10 to 30 minims to the ounce of water. Of these, carbolic acid is the most useful in the acute stage of eczema which we are now considering. All of these are used by dabbing them upon the surface for ten to twenty minutes, according to the relief which they afford, and then covering the surface with a soothing ointment. Their use is to be regulated according to the relief afforded, and to be repeated as itching recurs. An excellent plan of treatment of this sort in acute eczemas is that recommended by J. C. White: the parts are dabbed for fifteen to twenty minutes with black wash, full or half strength; the sediment is allowed to settle upon the surface, and after the application the surface is covered with zinc ointment spread on cloths. The applications of the black wash are made at intervals of eight to twelve hours. Sometimes surprising relief is afforded even in acute cases by dabbing the surface for a few minutes with irritating antiseptic solutions, such as bichloride solution, 1:2,000 or 1:3,000, potassium permanganate solution, 1:500 or 1:1,000; but these applications are risky. Special attention should be called to the application of hot water for its antipruritic effect. This may be plain or containing 20 grains of bicarbonate of soda to the pint. Cloths wrung out of water as hot as can be borne are repeatedly applied to the surface for five to fifteen minutes, and then followed by a bland ointment. For the relief of itching, especially in eczema about the genitals, it is a most valuable application, and, when followed by the immediate application of ointments, is beneficial to the inflammatory process as well.

Where the weeping is slight or absent a good form of lotion is one containing an inert or slightly astringent powder in suspension, which settles upon the surface when the lotion is dabbed on; the most useful of these applications is calamine lotion, in which there is one half to two per cent carbolic acid. Calamine, zinc oxide, and other inert powders may be added to such lotions as lead and opium wash. These lotions containing powders are particularly soothing in the dry forms of eczema where there is not too much weeping. In connection with these lotions, or in place of them, we use the various soothing ointments, among which especially to be recommended are rose ointment, and the other cooling ointments of the type suggested by Unna, zinc ointment, and diachylon ointment. To these may be added small quantities of the various antipruritics suggested as additions to the lotions; one half to one per cent carbolic acid is a good addition, or ten per cent of spirits of camphor, or six to ten per cent of boric acid. The addition of about one dram to the ounce of zinc oxide or zinc carbonate or bismuth subnitrate or other bland powders is usual in these ointments; they are slightly astringent and afford some additional protection, but are often, in my opinion, useless additions.

To get the best effect in acute eczemas an ointment should not simply be rubbed upon the surface, but should be spread in a thick layer upon soft, bleached gauze, and the spread ointment then smoothly applied to the surface. Under circumstances where such dressings are impracticable the ointments should be very abundantly applied—not rubbed in, but spread smoothly over the surface.

In place of ointments in acute eczemas a good substitute often is a paste, like Lassar's paste. Various drugs can be added to Lassar's paste. Lassar originally added two per cent of salicylic acid, but that is irritating to acute eczemas; one per cent of salicylic acid or carbolic acid is usually the best addition, if, indeed, the plain paste without any additional ingredients does not serve all of its purposes. Such a paste should be applied thickly over the surface and covered with gauze or absorbent cotton, or should be spread thickly upon gauze and then applied smoothly. In acute eczemas it should be changed two or three times in twenty-four hours. In less acute stages it can be worn for twenty-four hours or longer. Such a paste is liable to cake if it becomes dry, and may then be irritating. To remove it under these conditions, it should be softened with oil.

In cases where the eruption is very extensive, the use of lotions is not ordinarily satisfactory except for limited areas. In these cases we may rely at times upon the free application of ointments, on gauze where the condition is worst, and by simply greasing the skin elsewhere. In the place of ointments in extensive cases we may often use with advantage liniments, of which calamine liniment (*q. v.*) is a type and the most useful preparation. The entire affected surfaces are covered with cloths which have been wet in this liniment.

After an acute eczema is well on the road to cure, and in cases of dry erythematous eczemas, dusting powders may be sufficient and ar convenient and agreeable applications. They are of service also fo application over parts which are only slightly involved while we ar using ointments and pastes over other areas. The various dusting pow ders suggested under general therapeutics are used here, to which th commonest additions are powdered camphor, 10 to 20 grains to th ounce; salicylic acid, 5 to 10 grains; tar, 20 to 60 grains; salol, 5 to 1 grains; carbolic acid, 5 to 10 grains; boric acid, 1 dram to the ounce In the same way one can use a tragacanth cream or a tragacanth jell as a soothing application over surfaces showing a slight degree of eczema

With soothing applications, such as have been indicated above, w carry our cases through the acute forms of eczema. There may be som exacerbations to prevent the gradual subsidence of the process, but i spite of these one is usually able to cause improvement until the proce loses all of its acute features. It may be that these soothing measur will be all that is necessary in order to restore the skin to normal. I certain of the acute cases, however, and as a rule in the subacute an chronic forms of eczema, we have finally to resort to stimulating appl cations in order to get rid of the last results of the inflammatory proces If such applications are necessary in the course of recovery of an acut eczema, their use must be begun with great caution and increased tent tively in order to avoid exciting the disease anew. In chronic eczem also one must be cautious in the use of stimulating applications, but l may proceed with more freedom. There is no point in the treatment eczema where better judgment is required than in deciding when to pa from the use of soothing to stimulating applications in a subsidin eczema.

For stimulation in eczema tar is the most useful remedy. It ma be added either to ointments or in the form of compound tincture coal tar to lotions. In the beginning of its use after acute eczemas on should be very cautious, starting with 5 to 10 grains of tar or an equ quantity of compound tincture of coal tar to the ounce. If it agrees, the quantity can gradually be increased, until a dram or perhaps more is used to the ounce. Next in value as a stimulant are the salts of mercury, especially ammoniated mercury, beginning with 10 to 20 grains to the ounce. Instead of these we sometimes use carbolic acid, 10 to 20 grains to the ounce; sulphur, 10 to 40 grains; betanaphthol or salicylic acid, 30 grains; chrysarobin, 5 to 10 grains. These may be used in lotions, but are more frequently added to ointments. The following prescriptions are examples of good combinations for producing moderate stimulation:

℞ Sol. subacet. of lead	ʒss–i;
Comp. tinct. coal tar	ʒss–i;
Water q. s. ad	℥viij.

℞ Zinc sulphate, Potassium sulphuret, } āā	gr. v–x;
Water	℥iv.

℞ Pix liquida	♏x–xxx;
Zinc oxide	ʒi;
Rose ointment q. s. ad	℥i.

℞ Chrysarobin	gr. v–x;
Pix liquida	♏x;
Ammon. mercury	gr. x;
Benzoinated lard q. s. ad	℥i.

(After Hutchinson.)

The stimulating lotions are not applied constantly, but are dabbed upon the surfaces or the surfaces are bathed in them for from ten to thirty minutes, and then ointments used. The stimulating ointments are not usually applied in thick layers on dressings as are the soothing ointments, but their application is confined to rubbing them over the surface. Where the slightest effect is desired they are merely spread on lightly. The more they are rubbed in the more intense is the effect produced, and sometimes it is necessary to rub them into the surface for ten or fifteen minutes.

The use of stimulating applications is necessary, as a rule, only over circumscribed areas, for the inflammatory products that require absorption are not usually widely diffused. In treating comparatively small patches of chronic eczema with much secondary infiltration and scaling, we resort to much more vigorous plans of treatment, but under such circumstances we have to deal with distinctly chronic, low-grade inflammatory processes where the danger of acute eczema is remote. These vigorous stimulating methods of treatment, which are practically confined in their application to patches of chronic, indurated, and usually scaly eczemas, consist essentially in producing a short, acute inflammatory reaction, which is either allowed to subside of itself or is treated subsequently with soothing applications. A method of this sort is Hebra's soap treatment, which is very useful in the treatment of old thickened scaly patches of eczema. A lump of soft soap is rubbed thoroughly into the surface. Then the surface is rubbed with a cloth dipped in warm water until an abundant lather is produced; this is rinsed off and the surface dried. In this procedure the loose thick scales of epidermis are cleaned off, and slight oozing from the surface is produced. Immediately after drying, diachylon ointment, spread thick upon gauze, is applied to the surface. This procedure is repeated once or twice daily until all infiltration is gotten rid of. A plan of getting rid of thickened layers of horny epidermis, which can be carried out only by the physician, is to wash them off with a swab of liquor potassæ. This is rubbed over the surface until the thickened, horny layers melt away and punctate oozing occurs, and then diachylon or other soothing ointment is applied. This procedure can be repeated as soon as the reaction subsides. The same thing can be done by the application of salicylic-acid plasters, twenty to twenty-five per cent. These are applied for several days, until the horny epidermis becomes spongy and softened, and can be washed off. Salicylic acid, four to ten per cent, in flexible collodion, can be painted on the surface, and renewed from time to time until the same effect is produced. One of the best forms for employing salicylic acid in this way is in a soap plaster of the following formula:

℞ Emplast. saponis	90.0
Ol. olivæ	10.0
Acidi salicylici	5.0 to 20.0

This plaster should be spread upon muslin, or preferably upon thin chamois skin or kid, and the dressing then applied smoothly to the surface.

The salicylic-acid and the caustic-potash mixtures are indicated where we want to get rid of thickened layers of horny epidermis. If stimulation alone is desired without the removal of an excess of horny epidermis, the soap treatment is useful. Another particularly useful remedy is tar, in varying strengths, mixed with ointments or plasters, or in some cases even used pure and briskly rubbed into the surface. Occasionally, in an indurated patch, a good method of stimulation is by painting with silver nitrate, from 10 to 15 grains to the ounce up to the saturated solution in water or sweet spirits of nitre. To relieve itching, and as a stimulant, painting with potassium permanganate solution, from one or two per cent to ten per cent in water, is often valuable (Bulkley). A plan of treatment recommended by Crocker in chronic local eczemas is to soak the surface in oil overnight, wash thoroughly the next morning with bicarbonate of soda, 1 dram to water 4 ounces, and then, after drying the surface, paint with a ten-per-cent aqueous solution of potassium permanganate. This is repeated once a day until a black crust is formed, which is left for a week to exfoliate. The occasional painting with a strong solution of potassium permanganate is often an excellent plan of stimulation. Occasionally it is well to paint chronic patches with tincture of iodine, or even with Cutler's fluid (tincture of iodine, carbolic acid, and chloral, equal parts). The relief from itching which these various stimulating applications often afford is remarkable. It must be remembered that all these vigorous stimulating plans of treatment are applicable only to circumscribed areas, and require caution in their use.

In the treatment of subacute circumscribed patches of eczema, convenient and very efficient forms of treatment are found in the use of dressings which afford a maximum amount of protection and require infrequent changing. The plaster mulls of Unna are satisfactory for this purpose, but are difficult to obtain fresh and in proper combinations. A still better form of application is soap plaster containing ten per cent of olive oil, to which may be added salicylic acid, carbolic acid, tar, sulphur, as indications may require. The best form of fixed dressings for patches of subacute eczema is Unna's glycerine jelly. This forms a smooth, protective dressing which allows the evaporation of secretions. It is soothing and agreeable, and adheres well for several days. It may even be used where the process is rather acute, with some free exudation, and is a most useful application in eczemas of the legs, and in circumscribed patches where a permanent dressing is desired.

Special Forms of Eczema.—Certain forms of eczema, either because of their location or because of special features, warrant brief separate consideration.

Toxic Eczemas.—The eczemas of toxic and metabolic origin are usually symmetrical and affect by predilection the face and hands and feet, parts upon which the vasomotor control is most sensitive, and in considering the eczemas of these parts we have in mind especially eczemas of internal origin. These are usually only the points of greatest intensity, around which the eruption appears in greater or less abundance.

Perhaps the most characteristic form of regional eczema of internal origin is infantile eczema, and it may therefore well be considered first.

Infantile Eczema.[1]—This is a characteristic form of eczema which occurs in infants most frequently during the first and second years. Its typical location is over the flush area of the cheeks. In its mildest forms it may consist of only a slight scaly erythematous patch, or several patches, on either cheek, on which there may or may not be discrete papules or papulo-vesicles. In a little more intense stage it shows on the cheeks as coin-sized patches of papular or vesicular eczema surrounded by erythematous halos. It is but a step from this to well-marked pustular eczema of the cheeks, and the disease very frequently appears in this form. In such cases there is an acute dermatitis; the surface weeps profusely; as a result of secondary infection, there is an abundant formation of pus, which dries on the surface into yellowish dirty crusts.

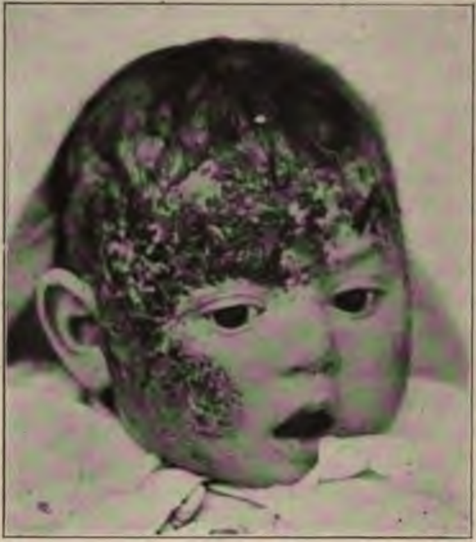

Fig. 78.—Infantile Eczema. Crusted pustular type. (Schamberg's collection.)

In the slightest cases of infantile eczema the eruption may be confined to the cheeks, but there are frequently a few vagrant lesions over different parts of the body. In the severer pustular forms the disease is likely to spread widely. In addition to the cheeks, it commonly affects the tip of the chin, the forehead, the folds behind the ears, the scalp, and the neck. Between the sites of greatest intensity on the head and face, there are apt to be numerous lesions which may be discrete, but in severe cases the disease may spread with greater or less intensity over the entire face, head, and neck. At the same time it is likely to appear around the wrists, on the legs, over the buttocks, and in the inguinal folds, and the eruption may be in the form of generalized discrete lesions. In the exaggerated cases the eruption will be weeping and raw or pustular at the points of greatest intensity. At points where it is less intense there will be discrete vesicles and papules occurring on erythematous patches or discrete lesions without any grouping. In all degrees of the disease it is not uncommon for urticarial lesions to occur irregularly over the body. In its exaggerated forms the disease presents a very disfiguring eruption, one which causes great distress to the parents, and it looks as though there might be left permanent scarring, which, however, is never the case. It pursues a very irregular course, getting better, perhaps entirely disappearing, and then recurring. In this way it may go on for many months.

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[1] Hall, *Brit. Jour. Derm.*, 1905, pp. 161, 203, 241, 287 (important paper).—*Ibid.*, 1908, p. 4.

The itching which accompanies this eczema is fairly severe, and causes constant scratching, which has to be controlled in order to prevent exaggeration of the disease. The patients, however, manifest little evidence of distress. They go on eating vigorously and thriving.

As a rule infantile eczema is unassociated with constitutional symptoms, but very rarely septic infection has its origin in suppurating infantile eczema. I have seen this happen in a child, with a pustular eczema of the face matted under crusts, who developed cellulitis in the neck, followed by a fatal septicemia. Hutinel and Rivet have described nine cases, and refer to others in French literature in which fatal septicemia has occurred in hospitals in children who were suffering from facial eczema.

ETIOLOGY.—This type of infantile eczema arises, in the vast majority of cases, from intoxication from the intestinal tract. It may occur in children who are poorly nourished and cachectic, but it is quite as frequent in chubby, fat babies who eat too much and suffer from gastrointestinal disturbances. It is most frequent in bottle-fed babies, but it also occurs in babies nursed at the breast. It is very common in babies who are constipated. Frequently in these cases the mothers are constipated, and there seems to be a reasonable assumption that toxæmia is in such cases secondary, for relieving the digestive disturbances, especially the constipation, of the mothers is often necessary before the nursing child will escape from the eruption. In other cases with no definite gastrointestinal disturbances the etiology seems to be the same as that of toxic eczemas in plethoric adults. These cases are usually seen in children a little older—from two to five years. They are vigorous, hearty children, who eat more than they can assimilate, are very fond of sugar and sweets, perhaps drink too little water, and are, in short, overwell nourished. It is only fair to say that some excellent authorities regard infantile eczema as of purely local origin. However, its distribution, its association with urticaria, and its rebelliousness if the gastrointestinal tract is not looked after, indicate strongly that it is of internal origin, and this is the opinion of most authorities. There are a good many cases which are chiefly confined to the face and scalp, in which, with an eczema on the face, there is a dry seborrhœa of the scalp, and possibly such cases are manifestations of seborrhœic dermatitis. But the treatment of the gastrointestinal tract is an essential of success in their management, and the local treatment does not differ materially from that of the toxic forms.

TREATMENT.—The treatment of this form of eczema requires in the first place careful attention to the gastrointestinal disturbances. If bottle-fed, the feeding has to be regulated carefully according to approved methods. As a rule, the milk has to be diluted more largely than has been done, and the amount of sugar cut down, and in well-nourished babies it is usually desirable to reduce somewhat the quantity of food. Where the child is being allowed more or less of sweets and unsuitable foods, these have to be rigorously cut off. In addition to such measures, aperients are in most cases needed. At the start it is well to repeat several times, at intervals of a few days, aperient doses of calomel or of gray powder. For daily use, as an aperient I have gotten excellent service from extract of malt, with enough of one of the tasteless preparations of cascara to keep the bowels well open. But the medicinal treatment of digestive disturbances in these cases is in no way peculiar. The chief point to be emphasized is the necessity for getting rid of the intestinal fermentation and keeping the bowels well open.

In the pustular forms, the first object is to clean the surfaces of the pus and crusts. This may be done by wet dressings of boric acid or by the application on cloths of bland oil or of a thick coating of vaseline, to which often may well be added one-half per cent of carbolic acid, or two per cent of ammoniated mercury, or six per cent of boric acid. After the surfaces are cleaned, antiseptic ointments, with vaseline or diachylon or rose ointment as a base, and containing carbolic acid one half per cent, boric acid six per cent, aristol twelve per cent, or any other of the mild antiseptics, are applied freely on dressings. On the face it is often convenient to apply the ointment under a mask cut out of muslin, in which there are holes for the eyes, nose, and mouth. When the suppuration has been stopped, any of these mild ointments may be used freely, and after the weeping has in great part stopped they may be used in conjunction with calamine lotion or with black wash after the manner already indicated. As the disease becomes still less active, it may be sufficient to use a lotion alone, like calamine lotion, or to apply the ointment upon the surfaces without covering.

In all cases except the mildest it is necessary to take measures to prevent damage from scratching. The hands should be muffled in gauze, and in extreme cases much is gained by putting the child in a sort of strait-jacket. He may be put in night drawers which are closed at the hands and feet; these are then pinned to the mattress in such a way as to prevent free movement of the arms and legs. A dressing can be extemporized by putting the child into a pillow slip, which is fastened by pinning to the mattress in such a way that the arms and legs are held fast. A child will submit to these jackets with no resistance after a short time, and they add very much to its comfort. If this is not done, a vigorous child will require the undivided attention of at least one person day and night to keep it from scratching.

Eczema of the Face in Adults.—Eczema of the face in adults usually occurs as erythematous or papulo-erythematous eczema, with more or less vesiculation. It is very rarely pustular. The patches occur by preference on the cheeks, nose, mouth, and forehead, or the eruption may be confluent over the face. In adults it is much more frequently a disease of local origin than in infants, but it is occasionally a manifestation of an internal disturbance. Of course, generalized eczemas and eczemas of external origin frequently involve the face, but these cases present no peculiar features, and require no especial consideration.

There is a certain type of eczema of the face in adults which is very rebellious, and is distinctly of vasomotor origin. This may occur as attacks of vesicular eczema which flare up, last for a few days, and go down, leaving perhaps slight induration or erythema or perhaps no trace during intermissions. In its most rebellious form it occurs as an erythematous eczema over the entire face, with more or less permanent infiltration of the tissues. On this, when exacerbations occur, as they habitually do, papules and vesicles develop in greater or less abundance. In the pronounced cases of this sort the skin may be permanently thickened with great exaggeration of its furrows, so that there is produced a pseudo-leonine face superficially resembling that of leprosy. This type of eczema of the face may be unaccompanied by eczema on other parts. The itching during exacerbations is almost intolerable and makes life a burden.

The papular and vesicular types of this form of eczema I have seen most frequently in women. The erythematous type, with great thickening of the skin and exaggeration of the furrows, has in my experience occurred most frequently in oldish men. This form of eczema may occur where no metabolic or toxic disturbance can be discovered. In some cases careful observation extending over years has not been able to determine any definite disturbance of health. The disease seems to be some deep-seated vasomotor disturbance, and the cases are most rebellious; some of them are totally beyond radical relief. The disease runs a varying course, with exacerbations and remissions, and perhaps intermissions, but obstinately recurs.

The local medicinal treatment is by the usual soothing and antipruritic remedies, which can be used in rather strong forms. In some of the cases I have given considerable relief by the mild use of x-rays, but have not been able to cure them. Counterirritation over the back of the neck should be tried, and every defect of health sought for, and if possible remedied. The patients may be improved by residence in a mild, equable climate.

Eczema of the Hands.—The hands are frequently involved in eczemas which result from local irritation. Eczemas of internal origin, especially eczemas due to metabolic and to toxic disturbances, very frequently involve the hands. Such internal disturbances may be the predisposing causes of eczemas which are excited by the local irritation of occupations, or in other cases these internal disturbances may be the exciting causes of the disease.

As the disease occurs on the back of the hands and the fingers, it usually appears as rather sharply defined groups of papular or papulo vesicular lesions on a sharply infiltrated erythematous base. The erup tion varies from time to time, disappearing perhaps entirely, and reap pearing, but is usually obstinate. Eczema on the palms usually occur in the form of ill-defined scaly patches of a pinkish color, and with good deal of infiltration. These patches may involve only part of th hands, and may be of ill-defined outline or have sharp borders. In othe cases the disease spreads continuously over the palmar surface of th hand, and may also spread, in the form of papular and vesicular lesion to the dorsal surface. At times the infiltration is very slight, an the entire palmar surface may be slightly thickened, of a delicate pin color, and with an exaggeration of the normal lines and furrow Where the infiltration is thick, the skin becomes inelastic, the fu rows are much exaggerated, and fissures occur as a painful compl cation of the condition. Eczema of the hands of internal origin usually symmetrical, and is likely also to involve the feet. This sym metry is an important diagnostic feature between it and the chroni scaly syphilides of the palm of late syphilis. Itching is usually presen but merely amounts in most cases to an annoyance.

Eczema of the hands is rebellious, and may persist for years.

TREATMENT.—In the first place, all local causes must be sought f carefully, and as far as possible eliminated. The patient's diet an manner of living are usually at fault in the persistent cases, and shoul if possible, be corrected. In this form of eczema especially we have use measures to overcome the so-called gouty and rheumatic diathes and disturbances of digestion.

In acute stages, local treatment consists in using soothing measure In the chronic types, stimulating forms of treatment are necessary, lik the soap treatment of Hebra, and the use of salicylic-acid plasters. I the chronic scaly patches on the palms, one of the best methods treatment is the use of soap plaster 90 parts, and olive oil 10 part to which is added from fifteen to twenty-five per cent salicylic acid. This is applied in a thick layer on gauze, and is left on until the horny epidermis becomes soft and loose, and can be easily removed. After cleaning off the softened horny masses, a bland ointment is used until the reaction subsides, and then the keratolytic application is again used.

A plan of treatment of eczema of the palms recommended by Jamieson is as follows: After softening of the thickened epidermis with starch-jelly poultices in which boric acid has been incorporated, changed every four or six hours, the skin being rubbed briskly between times with a rough, soft, dry cloth, the following is to be rubbed in sparingly each day:

℞ Acidi pyrogallici oxidati	gr. v–xxx;
Lanolini	℥ss;
Aq. destillatæ, Ol. amygdalæ, āā	ʒij.

Eczema of the hands occurring as erythematous patches, or patches of papules or vesicles on erythematous bases, is not uncommon in nurses and surgeons from the habitual use of antiseptics. In these cases much can be done by exercising great care in drying the hands thoroughly, and by the subsequent regular softening of the skin with a one- or two-per-cent tragacanth cream or a bland ointment. Suitable ointments are first diachylon, then rose ointment, vaseline, lanolin, or various mixtures of these. The habit of lightly anointing the hands with neutral ointments after washing will do much to prevent such eczemas. The tragacanth cream can be used for the same purpose; it is a little less efficient, but somewhat more agreeable.

In chronic eczema of the hands, I have found the application of x-rays beneficial in many cases.

Eczema of the Nails.—The matrix of the nails may be involved in eczemas of the hands. It shows in more or less interference with the nutrition of the nail plate, in loosening of the nails, and in the accumulation of horny material under the free borders. It is very difficult to cure. The thickened horny mass under the edge of the nails may be dissolved out by the use of a thin swab of cotton dipped in liquor potassæ, and stimulating ointments may then be worked up under the nails. The same thing can be done by working up under the nails salicylic-acid ointment in strength of five to ten per cent. Whatever the treatment pursued, the stimulating applications have to be used intermittently but persistently under the borders of the nails, and every effort made to overcome the underlying causes.

Eczema of the Feet.—Eczema of the feet occurs frequently in association with eczema of the hands, and usually in such cases it occurs in the same form. It occurs on the dorsal surfaces of the feet as patches of papular or vesicular eczema, and on the soles as indurated scaly patches. The lesions on the soles frequently become much more thickened and hypertrophied than the corresponding lesions on the palms, because of the greater pressure and friction to which they are exposed. Except for this difference, the lesions on the soles are similar to those on the palms, and they require the same sort of treatment; the use of keratolytic applications, to get rid of the excess of horny epidermis, or the application of stimulating remedies, followed by soothing ointments Chronic eczema of the soles is rebellious to treatment, and is at time very troublesome.

The papular and vesicular types occurring in patches on the dors surfaces of the feet are quite analogous to the same forms of eczem occurring on the backs of the hands and require the same sort treatment.

Eczema on the feet, especially between the toes, frequently occu as the result of poor circulation and hyperidrosis. In addition to t ordinary methods of treatment of such eczemas, these cases are like to be greatly benefited by the use of astringent washes. Solution subacetate of lead and tincture of opium, ½ dram to 1 dram each to t ounce of water, is an excellent application in such cases. The f may be bathed in this twice a day, or wet dressings may be put during the night. Where irritation is not marked, washing the feet tannic-acid solution ten per cent is useful, or freely dusting the f with boric acid before the stockings are put on. (Cf. Hyperidro Pedum.)

Eczema of the Legs.—Eczema of the legs is a persistent and v common form of the disease. An important factor in its production the venous stasis which results from the obstacle that gravity prese to the return of the blood from the legs. The condition, therefore, exceedingly common as a sequel of varicose veins of the legs. Ecze of the legs is usually symmetrical, and its sites of greatest intens are, as a rule, the sides and backs of the legs between the ankle a the middle of the calf. There occur all degrees of papular and vesicu eczema from a few grouped lesions to large weeping areas involvi almost the entire leg. When the lesions are few they are usually loca on the sides and back of the leg, above and around the ankle, and the sides of the heel. There may be only one or two small patches there may be large areas entirely denuded of epidermis, of blood- color, weeping profusely, and with more or less suppuration. The co of these areas is usually blood-red, but in some cases it may be a disti purple. These extensive weeping eczemas are the typical forms of ecze rubrum. As a result of long continuance of the process, secondary mmatory changes are common. Great thickening and induration of e skin frequently occurs, and ulcers may develop, especially on the les of the legs above the ankles. These ulcers are usually round, arply circumscribed, and with thickened, sometimes almost cartilagous, borders which require scarification before they can be made to solve. In some cases there occurs great hypertrophy of the con-

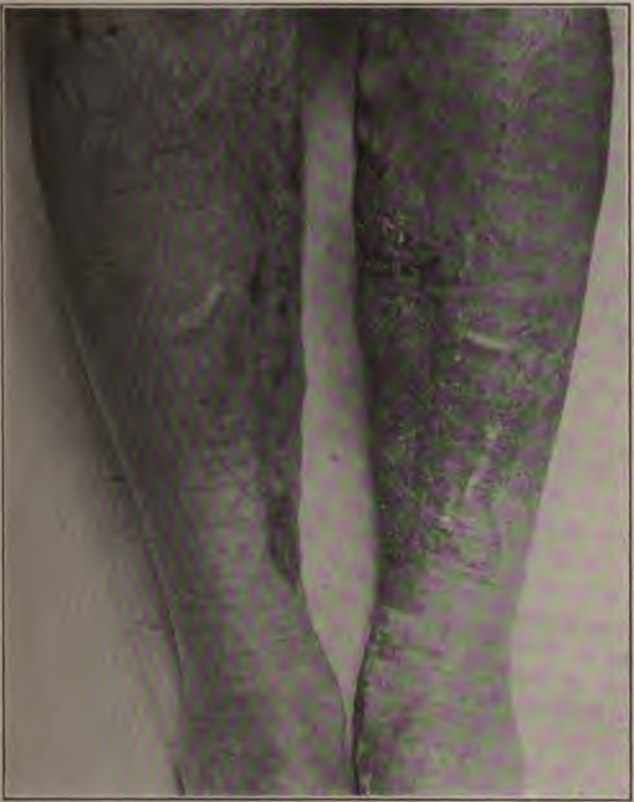

FIG. 79.—ECZEMA OF LEGS WITH VARICOSE VEINS. (Author's collection.)

ctive tissue, and even elephantiasis. In these cases epithelial hyperphy is often also pronounced, showing as wartlike, rough, horny rgrowths.

The TREATMENT of acute eczemas on the legs is like that of similar emas on other parts, with the addition that provision usually must made for the support of the venous circulation. In very acute cases may use wet dressings of boric-acid or lead-and-opium wash or aluum-acetate solution, or, in the place of these, bland ointments, and, he process subsides, ointments are preferable. The ointments should be spread on gauze, and, whatever form of treatment is used, the should be supported by well-applied bandages from the toes to below the knees. In acute cases the patients do best in bed.

In subacute eczemas of the legs without much exudation, one o best applications is Unna's glycerine jelly. This may be applied the ulcer in some cases; in others, an opening may be made i

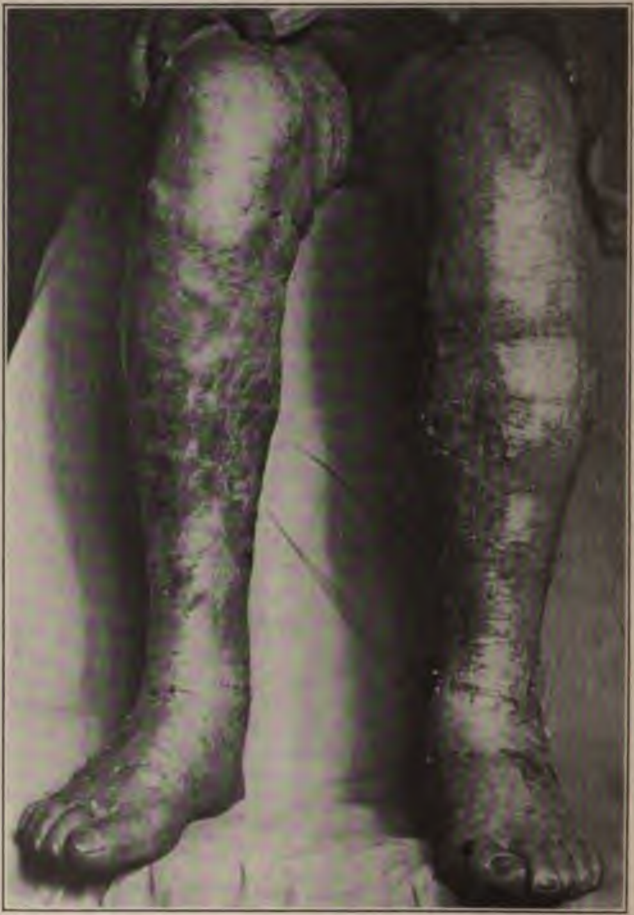

FIG. 80.—ECZEMA RUBRUM OF LEGS. In nature the skin was blood-red, te glistening, with little weeping. (Author's collection.)

dressing over the ulcer, and a dry dressing applied there. Unna' is a very useful application in cases requiring only slight support, the use of a bandage is to be obviated.

In the chronic infiltrated cases stimulating applications may be but their use requires caution. For the ulcers, wet dressings or ointments may be used, applied under supporting bandages. For the relief of pain of the ulcers, lead-and-opium wash in wet dressings is frequently sufficient. Sometimes it is necessary to paint the surface with cocaine or eucaine, two to four per cent twice daily. In place of this, cocaine ointment, one or two per cent, may be applied, or orthoform may be lightly dusted over the ulcers before the application of an ointment dressing. Orthoform, however, has to be used cautiously, for it frequently sets up a dermatitis, and I have seen it make ulcers of the leg gangrenous. For the treatment of the thickened, dense border of some of the ulcers, deep scarifications through the border, repeated at intervals of about a quarter of an inch around the entire ulcer, are very useful.

Eczema of the Genitals and Anus.—The genitals and the anus and the contiguous skin in both males and females are frequent sites of most rebellious eczemas, and they are usually very distressing from the severe itching that accompanies them. This is especially true of the eczemas of the vulva and anus. As a result of long persistence, the skin becomes infiltrated, inelastic, and fissured, and upon this thickened skin exacerbations of acute eczema come and go.

These chronic rebellious forms of eczema about the genitals and anus are sometimes associated with some constitutional disturbance. It may be metabolic or toxic, but the cases usually occur in patients of unstable or depressed nervous systems.

There is no form of eczema more difficult to control. In the TREATMENT of the cases, the first thing should be to discover, if possible, the underlying causative factors. Nutrition and elimination should be carefully looked into, a bland diet should be ordered, highly seasoned foods prohibited, and tobacco and alcohol and other stimulants, if possible, cut off. The urine should be watched especially for sugar. The fissures which occur in anal eczema frequently cause its continuance, and they should be treated either by linear scarification, painting with nitrate of silver, or dilatation of the anus under anæsthesia. Where the eczemas are acute, they should be treated with soothing applications until the active stage is passed. For this purpose powders are usually best, for ointments, unless lightly applied, are not ordinarily well borne on these parts. Where the conditions are chronic, or after acute exacerbations have passed, strong antipruritic applications are indicated, and of these carbolic acid is most frequently beneficial. In the agonizing waves of itching of the vulva and anus, great relief may be afforded by painting the surfaces with one- or two-per-cent solution of cocaine or eucaine. At other times, painting with solution of potassium permanganate, one-half to one per cent, gives relief, and is positively beneficial to the condition. Better than either of these in relieving itching is the use of water as hot as can be borne, applied repeatedly for ten or fifteen minutes at time, and followed by a soothing ointment. This is one of the mos useful measures in such cases; it not only relieves itching, but improve the condition.

In these chronic cases of eczema ani et vulvæ many practitioner have found the careful use of x-rays of great service, and this agen has given me very satisfactory results.

These persistent chronic forms of eczema about the genitals ar quite independent of and quite different from acute eczemas whic frequently develop in these places. In acute eczemas of these part local irritation must especially be looked out for—parasites, irritatin secretions, and the effects of warmth and moisture and friction of th parts. Such cases are treated in the usual ways with soothing, mil antiseptic applications, and offer little difficulty in treatment. As a ready remarked, powders and lotions are better borne here than oin ments, but in some cases ointments must be used, and then it has t be seen to carefully that they are thoroughly applied. This is partic larly true of eczema of the scrotum, where ointments should be applie on dressings carefully adjusted.

Eczema of the Bearded Part of the Face.—Pustular eczema of th bearded part of the face is very common, and differs from sycosis (pu tular folliculitis) in the readiness with which it yields to mild ant septic applications. One of the best applications is ammoniated me cury, 10 to 40 grains to the ounce of vaseline or rose ointment. Th surface is first cleansed by wet dressings of boric acid or by thorough washing with a mild antiseptic solution, and the ointment is then a plied on gauze, or is spread over the surface as frequently as is nece sary. After horny epidermis has formed and a slight erythematou eczema is left, a mild ointment may be used, or calamine lotion tragacanth cream.

Eczema of the Eyelids.—Eczema of the eyelids, or blepharitis, is n infrequent in poorly nourished children. The crusts should be remov by softening in oil, and after that a weak antiseptic salve should applied. Usually the condition yields readily upon the frequent appl cation of a salve of 4 grains of yellow oxide of mercury to the oun of vaseline.

Eczema of the Ears.—The folds behind the ears and the auricl are common sites for ordinary forms of eczema, and they are treat in the usual ways. The auricles are also a frequent site of seborrhœ dermatitis, and in eczema of the auricle seborrhœic dermatitis mu always be borne in mind. When it exists it requires the same treatme as seborrhœic eczemas in other locations, especially by applications sulphur and ammoniated mercury.

Eczema of the Lips.—Eczema of the lips may be confined within the vermilion border or spread beyond it. The lips are tender and the mucous membrane is dry, crinkled, inelastic, and furrowed. In severe cases the lips are more or less raw from the formation of fissures and from the pulling loose of the epidermis. Beyond the vermilion border the eczema shows as red patches of erythematous or weeping eczema of the ordinary type.

Eczema of the lips may be produced by seborrhœic dermatitis. It is not infrequently excited by exposure to drying winds, especially if the patient has the habit of constantly moistening the lips. It may also supervene upon abrasions of the lips, and especially upon the bases of lesions of herpes. It may be sometimes excited by internal causes; it may be associated with gastrointestinal disturbances, and it is frequent in nursing women, in whom it persists as long as nursing is continued.

Whatever produces it, the patients should be cautioned against the habit of moistening the lips, and the lips should be softened with a bland ointment as often as they become dry; this is especially necessary before retiring. As the trouble is so often a form of seborrhœic dermatitis, sulphur in nonirritating proportions (10 to 30 grains to the ounce) is usually a good addition to the ointment. The fissures may be cured by simply keeping the lips well greased. If this fails, they frequently get well under applications, at intervals of a few hours, of tincture of benzoin, which acts chiefly as a protective. Where the fissures are deep and are constantly broken open by the movements of the mouth, they can be cured within a day or two by painting them with plain collodion, which simply serves to immobilize the fissures and prevents cracking. The collodion should be painted up to the moist border of the lip, and should be renewed as soon as it loosens.

Eczema of the Nipples.—Subacute eczema of the nipples frequently occurs in nursing mothers. The dermatitis in these cases can be reduced by the ordinary soothing applications. The nipples should be thoroughly dried after nursing, and protected with a bland ointment, to which, in these cases, a good addition is carbolic acid (one per cent) or calomel or ammoniated mercury (two to five per cent). It is well in these cases for the ointments to be fairly firm. Diachylon ointment is a good base, or vaseline or cold cream may be used with the addition of 2 drams of zinc oxide or 1 dram magnesium carbonate to the ounce. These cases are usually made distressing and are kept up by the fissures which occur. These may be painted with compound tincture of benzoin, which affords good protection, or, better, they can be brought together by painting them with collodion. Frequently they need painting with nitrate of silver.

The nipples are frequently the site of eczema in scabies, but othe characteristic sites of scabies will also be affected.

Persistent indurated eczema, sharply circumscribed and involving th nipple and areola, especially if unilateral, should excite suspicion o Paget's disease (*q. v.*).

II.—VARIOUS FORMS OF DERMATITIS

Here are described various conditions which are plainly due to e ternal irritants. The lesions may be indistinguishable from those th have been described under eczema, and indeed the whole eruption often indistinguishable from an ordinary eczema. In some of the co ditions, however, the inflammatory process oversteps the bounds with which our conceptions have limited eczema. Thus, in dermatitis fr ivy-poisoning, there is frequently a violent reaction, with great swelli and with the formation of large bullæ. Again, the irritants may be intense as to produce necrosis; as, for example, occasionally from c bolic acid. Usually dermatitis from external irritants has characterist which distinguish it, such as location, outline, and frequently a shar defined border corresponding exactly with the extent of the irritant

Erythema Simplex[1]

SYNONYM: *Dermatitis simplex.*

Erythema simplex is a simple dermatitis excited by irritants of m erate intensity, and is so named because it is characterized by hyperæ redness. It corresponds to the mildest forms of erythematous ecze and would be better called dermatitis simplex. It occurs in patc which vary in their shade of red according to the intensity of the pro and in shape according to the extent of the action of the produ irritant. It may not pass beyond the stage of a dry erythematous der titis, but frequently develops into a superficial vesicular dermatitis. is accompanied by slight tenderness and burning sensation, and mor less itching. Like other forms of dermatitis, it may develop:

(1) From mechanical irritation, like friction or pressure, as f clothing or from exercises.

(2) From the irritation of radiant energy, such as sunlight (*thema solare*, or sunburn), x-rays, and heat and cold (*dermatitis* *rica, erythema ab igne*).

(3) From chemical irritants, like poisonous plants and var irritating chemicals, such as drugs, and chemicals used in the (*dermatitis venenata*).

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[1] Cf. Erythema simplex, under Congestive erythemas.

Simple traumatic dermatitis, like chafing of the thighs from walking, or irritation of the neck from a rough collar-band, or of the palms from exercise, needs no description. Dermatitis calorica—ordinary burns from heat—belongs properly to surgery.

INTERTRIGO

SYNONYMS: *Erythema intertrigo, Eczema intertrigo.*

Intertrigo (*inter* and *terere*, to rub) is a superficial dermatitis which occurs in the folds of the body. It occurs in adults, especially in fat people, in the gluteal folds, between the scrotum and thighs, in the folds of pendulous breasts, and in the folds of the neck. It is seen most frequently in fat children, especially in the inguinal folds and around the genitals and anus. Here, in addition to the irritation produced by the accumulation of the secretions of the skin, there is often additional irritation from soiled napkins. In its first stages it appears as a dry erythematous dermatitis. When it becomes more acute, vesicles develop, which quickly rupture, leaving a weeping red surface. It is accompanied by some tenderness, and perhaps itching.

There have been some attempts to establish it as a specific disease of definite etiology. It is a simple inflammatory process excited by the irritation of friction and irritating, usually decomposing, secretions. Its characteristic features are its location, its development as a result of friction and moisture and perhaps decomposing secretions, its superficial inflammatory character, and its rapid disappearance upon the removal of the causes and local treatment. It is only likely to be confusing as it occurs in infants. Here it must be differentiated from the erythema of congenital syphilis. Intertrigo about the genitals and anus of infants corresponds in its distribution to the area covered by the napkin, and is not likely to be complicated by destruction of connective tissue. The erythema of congenital syphilis occurs on the buttocks and around the genitals, but spreads down upon the thighs and legs, at times as far as the feet. At the same time flat condylomata and mucous patches and ulcerative lesions are very likely to be present around the anus, and other lesions of syphilis over the body.

Care should be taken that the parts are kept clean and protected as far as possible from friction. The area should be washed twice or three times a day with a slightly astringent solution, such as boric-acid solution or one-half- to one-per-cent carbolic-acid or lysol solution. After this they should have bland protective applications. Ointments are not, as a rule, the best preparations for use in the folds of the body, and in intertrigo powders and lotions are usually most satisfactory. If the reaction is slight, with little or no discharge, a bland antiseptic powder, like boric acid ʒi, and talcum ℥i, or sterate of zinc ℥ss, frequently is sufficient. Under pendulous breasts, such powders, in Unna's powder bags, may be used. If there is a weeping surface, soothing astringent lotions applied as wet dressings may be used with advantage. Suitable mixtures are boric-acid solution, one- to two-per-cent solution of aluminum acetate or subacetate of lead. In some cases the bland antiseptic ointments act well, like oxide of zinc ointment with the addition of boric acid ℥ss, or carbolic acid, 2 to 5 grains to the ounce.

ERYTHEMA SOLARE

This is the familiar sunburn, and is usually a dry erythematous dermatitis. It may at times be vesicular or bullous, but never of itself causes ulceration. It is produced not by heat, but by the actinic effects of the rays at the violet end of the spectrum. There is no better way of treating it than by using calamine lotion, followed by a bland ointment like boric acid and rose ointment. Where very acute, it may b treated, until the vesicular stage is past, with wet dressings of boric-aci solution or aluminum-acetate solution or lead-and-opium wash.

ERYTHEMA AB IGNE

Erythema ab igne is a condition which is seen in persons like fire men, ironmakers, and bakers, who are habitually exposed to radiant heat Its usual location is upon the front of the legs, but it is sometimes foun on the forearms and elsewhere. It occurs as rings of erythema, varyin in size up to one and one-half inches or more in diameter. Joinin rings may coalesce to form gyrate figures. The rings are of a dark re color, with centers of a brownish or reddish-brown tint. Usually the are dry and without elevation, but in rare instances they are elevate and occasionally vesicles develop upon them. As the process subside the color changes to brown from the presence of residual blood pigmen in the tissues, and the pigmentation may remain for a long time or b permanent. The subjective sensations are nil or a slight feeling o tenderness and heat or itching. The chief importance attaching to th condition is the recognition of its character and cause. It requires n local treatment, but soothing applications, like calamine lotion or lini ment or a bland stiff ointment, are agreeable. Its radical relief demand avoidance of the exciting cause.

X-RAY DERMATITIS

X-ray dermatitis is due to the actinic properties of x-rays, just a sunburn is due to the actinic rays of light; and if this is borne in mind it is very easy to appreciate the various forms of x-ray dermatitis. Th difference between dermatitis produced by x-rays and that produced by sunlight is that the actinic effects of x-rays are very much stronger and, if exerted to the maximum, produce effects upon the skin far exceeding those produced by light.

X-rays produce stimulation of pigment, tanning the skin and bringing out freckles in the same way that sunlight does. As with sunlight, this may occur without dermatitis. Aside from pigmentation, the first manifestation of x-ray effect on the skin is superficial erythematous dermatitis, most marked around the follicles. It is attended by slight itching and burning. The process may stop at this point, subsidence being followed by slight desquamation and pigmentation. Going beyond this, it becomes a lively red dermatitis, which is at first dry, but is likely to go on to vesiculation, with perhaps the formation of bullæ from the confluence of vesicles. The vesicles and bullæ rupture, leaving a weeping surface. The subjective symptoms are like those of a similar degree of dermatitis from other causes. The process may subside from this point. If it goes beyond this, the surface becomes dark, angry red, with intense congestion, which it may be almost impossible to remove by pressure. This cyanosis may occur before vesiculation appears, or it may follow vesiculation. If it appears before vesiculation, vesicles and small bullæ then develop upon the surface and, rupturing, leave a weeping, congested, purplish area, surrounded by a deep inflammatory areola. There then develops upon this raw surface a thin, gray necrotic membrane. This is closely adherent, and its forcible removal is followed by free bleeding. This necrotic membrane is made up solely of epithelium. With burns of this degree there is likely to be considerable swelling of the connective tissue. There is a variable amount of burning and itching, and occasionally slight pain, but the pain is not comparable in severity with that of x-ray burns with destruction of connective tissue. Even these cyanotic burns usually subside without necrosis of the connective tissue. The yellowish necrotic membrane is thrown off, horny epidermis grows rapidly around the borders, islands of horny epidermis spring up over the surface, and in the course of three or four weeks the area heals. Occasionally this involution is interrupted by the reappearance of bullæ and vesicles, and the condition may then continue for weeks, or even months.

If the process goes beyond this stage there occurs necrosis of the corium and subcutaneous tissue, usually not going below the subcutaneous tissue, but in some cases involving everything down to bone, and even including that. The necrotic ulcer which then results is unique. There develops a hard, leathery, dark gray mass of mummified tissue, which is closely adherent and persistent, and surrounded by an inflamed border. It is associated with intense pain, which completely destroys the patient's rest and peace, and will in a short time make him a nervous wreck.

In all degrees of x-ray dermatitis, dermatitis may develop upon other parts of the body as with other forms of dermatitis.

After dry erythematous x-ray dermatitis, there may be left no permanent effects upon the skin. After the severer reaction, there are left more or less permanent alterations in the skin which correspond in degree with the intensity and the duration of the preceding dermatitis. In a general way these changes correspond to the senile changes in the skin. The most frequent after-effect is a slight atrophy of the skin; the skin is a little thinner than normal, and shows some exaggeration of the normal lines. This change may disappear in time. Where the changes are greater the skin is thin, smooth, and glistening, of a lighter color than normal, but of a mottled pinkish color which is made greater by the development of dilated blood vessels (telangiectases). The surface is roughened by the formation of hyperkeratoses; the hairs are lost or grow sparsely, and the nutrition of the nails is more or less affected if the burn has involved the hands. In the most exaggerated cases of chronic x-ray dermatitis, following severe burns or following long-continued exposures to x-rays, as in operators, epitheliomata have developed in the hyperkeratoses, just as they develop in senile keratoses.

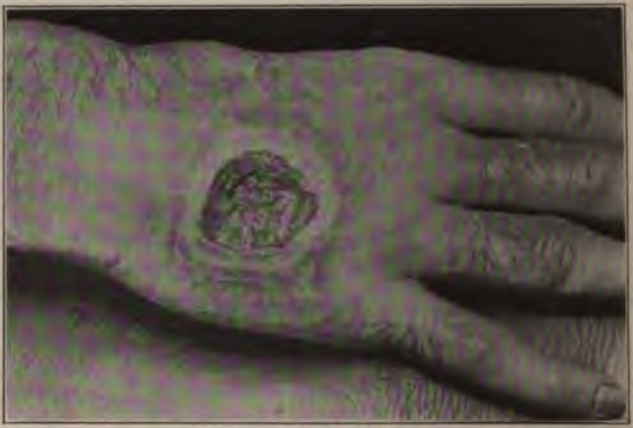
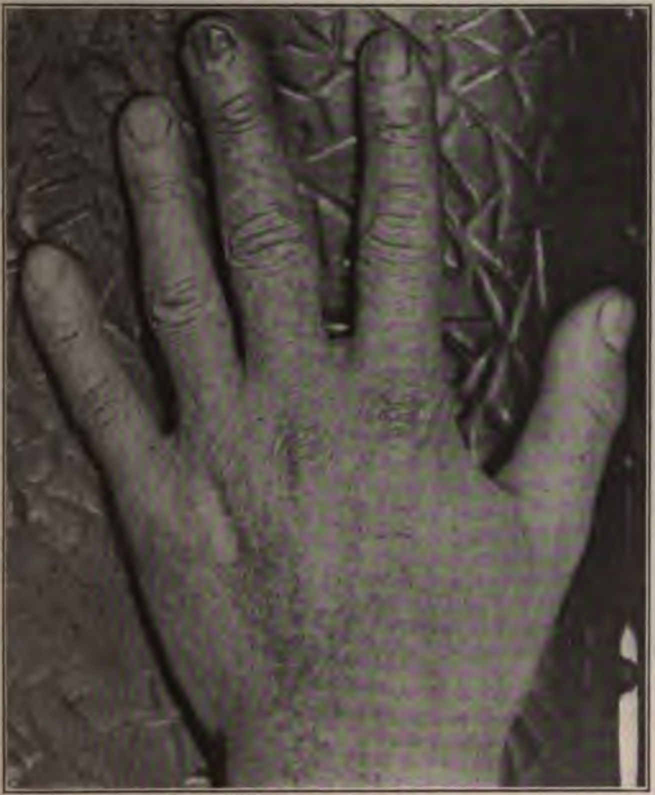


FIG. 81.—SMALL DEEP X-RAY BURN. (Author's collection.)

The histological changes in acute x-ray dermatitis are in the corium those of an inflammatory process with which there is endarteritis. The obliterating of many of the blood vessels as a result of the endarteritis accounts, in my opinion, for the permanent dilatation of the capillaries which results. In the epithelial structures there are marked degenerachanges, which cause destruction of the follicles of the skin, para- itosis, and ultimately atrophy of the epidermis.

The treatment of slight x-ray burns is like that of other similar ms of dermatitis. Where the surface is dry, a simple dusting powder better, calamine lotion does well. In acute vesicular dermatitis, wet ssings act well in most cases. I have generally used boric-acid wet ssings, but in some cases aluminum-acetate and lead-and-opium wash l various other bland wet dressings may serve better. In very sensi- cases, wet dressings of normal salt solution are least irritating. netimes these cases do better when covered by ointments on gauze, l for this purpose I have found nothing answers better than boric- l and rose ointment. In the sloughing burns everything can be l without avail, and the best course to pursue with them is, if possi- to excise them thoroughly and cover up the area by a plastic opera-



82.—Chronic X-ray Dermatitis of Hand in X-ray Operator, showing Crackling of Skin, Hyperkeratoses, Telangiectases, and Atrophic Nail Changes. (Author's collection.)

tion. In the chronic x-ray burns with telangiectases, hyperkeratoses, and an irritable dry skin, a good deal of comfort is gotten from the habitual frequent use of emollient ointments. For this purpose diachylon ointment is one of the best. In these cases Unna recommends hot bathing with decoction of arnica, and the subsequent application of an ointment, such as diachylon.

DERMATITIS VENENATA[1]

Under the title dermatitis venenata (venenatus—poisonous) are described various conditions due to substances chemically irritating. These are almost innumerable and include the various chemicals used in medicine and the arts—mustard, cantharides, chrysarobin, mercury, croton oil, resorcin, iodine, iodoform, orthoform, and other iodine compounds, benzine, kerosene, turpentine, potassium bichromate, metol, aniline, and arsenical dyestuffs—and many poisonous plants — rhus toxicodendron (poison ivy and poison oak), and other less common forms of the rhus species, rhus venenata (poison dogwood), poison sumac, urtica doica, primula obconica,[2] mucuna pruriens (cowhage), smartweed, oleander, euphorbia marginata (snow on the mountain), and rue.

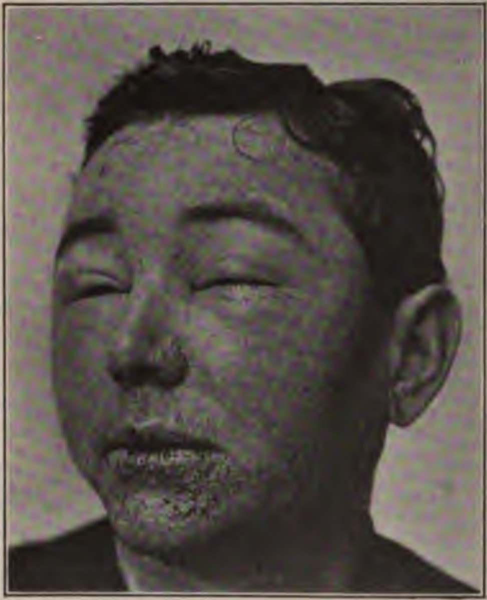


FIG. 83.—VESICULAR DERMATITIS VENENATA. (Schamberg's collection.)

Some of these substances, such as some of the mercurial salts, croton oil, or chrysarobin, will produce a dermatitis in any individual, but with most of them personal susceptibility plays an important part. This idiosyncrasy may be an unusual susceptibility to many irritating substances, or may be limited to a few.

Poison ivy may be taken as a type of these irritants. In the slightest cases it produces a dry erythematous dermatitis; most frequently it produces an acute vesicular dermatitis, and not infrequently a violent inflammatory reaction with the formation of large bullæ, and extensive, angry erysipelatous swelling of the parts. In very rare cases there may even be sloughing of the connective tissue. The parts usually affected are those exposed to its action—the hands or face and forearms. Often t involves parts which are not exposed, especially the skin around the genitals and anus, and it may be accompanied by widely diffused dermatitis, affecting especially the folds of the skin. The development of the dermatitis on parts which have not been exposed to the poison is sometimes explained by auto-transference of the poison from affected areas. The development of a widely diffused, symmetrical dermatitis, such as is not uncommon, cannot be satisfactorily explained in this way, but suggests strongly that its spread to unexposed parts is, at least in many cases, sympathetic. This is the more probable, because such sympathetic spread of a dermatitis to unexposed parts is a common occurrence with all forms of local dermatitis. Dermatitis from rhus poisoning may appear immediately after contact with the irritant, or it may develop in from a few hours to one or two days after exposure. It usually subsides in a week or ten days. Occasionally it is prolonged for several weeks, and sometimes as a sequel there are recurrent attacks of derma-

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[1] White, J. C., "Dermatitis Venenata," Boston, 1887; *Jour. Cutan. Dis.*, 1903.

[2] Nestler, "Hautreizende Primeln," Berlin, 1904 (Bibliography).

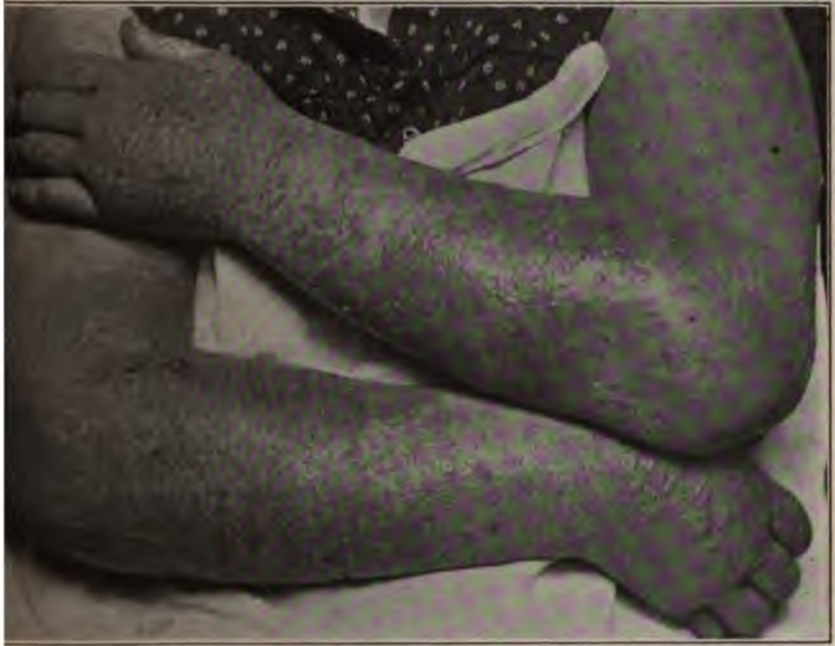

FIG. 84.—VESICULAR DERMATITIS FROM POISON IVY. (Author's collection.)

and removal of the oil. Another rational plan of treatment is the use of dilute subacetate-of-lead solution, the lead combining with the oil, but the precipitate must be removed from the skin, as it subsequently decomposes and frees the oil. I have lately found washing the parts with 50 per cent to 90 per cent alcohol of great use, even after they are virulently inflamed. It is not very painful and is followed promptly by great improvement. After washing, which may be repeated several times a day, the surfaces may be protected by a lotion, as calamin lotion, or a bland ointment, as rose ointment.

Along with the measures to get rid of the poison, or independently of them, local treatment, such as is applied to other forms of acute dermatitis, is usually effective, but ointments are not well borne.

FEIGNED ERUPTIONS[1]

SYNONYM: *Dermatitis artefacta.*

Dermatitis is occasionally produced with external irritants by patients. It may be any degree of dermatitis up to a sloughing ulcer. These lesions usually betray their production by certain artificial characteristics. They are likely not to conform to any ordinary type of lesions, to be of peculiar shape—quite round, linear, or angular—and with very sharp borders, and to be upon sites within easy reach of the right hand. One may even see the evidences of where the irritant has dropped on or trickled down over the surface. Artificial dermatitis is usually in the form of ulcers, the individuals repeatedly applying some form of irritant until ulceration takes place, and in many cases the lesions are covered by black, gangrenous sloughs. Occasionally the lesions are more superficial. The ulcers may be multiple or single, but there is practically no limit to the number of lesions which patients may inflict upon themselves. The history is usually one of repeated recurrence of one ulcer after another, continuing for months, and indeed, in some cases, for years. Scarring, of course, follows the deeper ulcers, and the patients may continue to inflict the lesions on themselves until they are greatly disfigured.

The cases are usually seen in emotional individuals, most frequently in hysterical girls, and occasionally in malingerers who want to use their disease as an excuse. It is possible that the condition sometimes occurs in patients with dual personality, who when in their normal personality are really unaware of the self-infliction of the injuries. A motive may be found in a desire to excite sympathy or to avoid duties, or it may be entirely undiscoverable. Any available escharotic may be used by these patients, but carbolic acid is the agent selected in most cases, probably for the reason that it is most familiar and most available.

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[1] Van Harlingen and Stout, Morrow's "System," p. 364.—Shephard, *Jour. Cut. and Gen.-Urin. Dis.*, 1897, p. 544.

In treating such a case it is not safe, until one has entirely gained the patient's confidence, to let him know one suspects the character of the dermatosis. The lesions should be treated according to ordinar principles, and covered with a dressing so that the patient cannot ge at them. If new lesions continue to appear, one may sometimes sto them by proper treatment, along with the hint that that will end th trouble if the patient lets them alone. If that is insufficient, the patien has to be watched until convincing proofs of the production of th lesions can be discovered, and then he should be confronted with th proof.

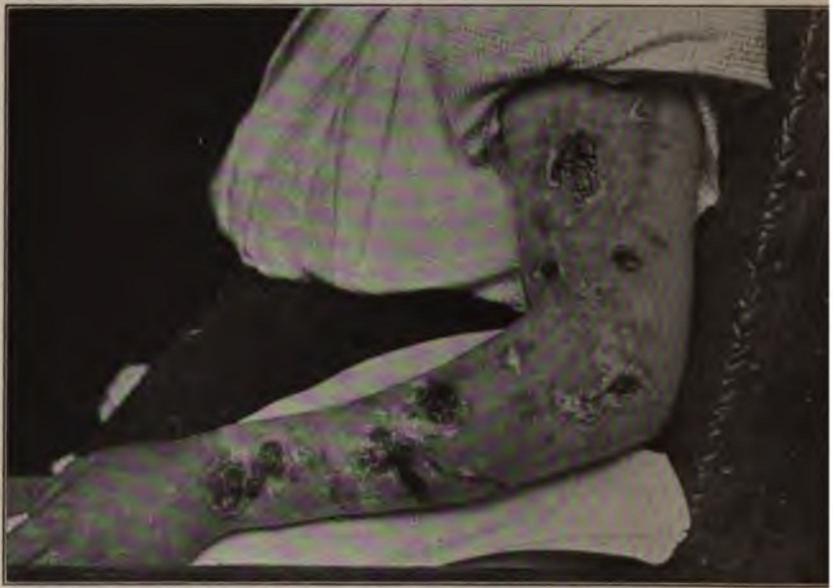

FIG. 86.—FACTITIOUS ERUPTION. (Author's collection.)

III.—GANGRENE OF THE SKIN[1]

SYNONYM: *Sphaceloderma* (σφάκελος—gangrene).

Gangrene of the skin shows the well-known features of gangrene i general. It may be moist or dry. It may begin with evidences of failur of circulation of the part, or appear suddenly without them, or follow various destructive processes without other circulatory disturbances than those of inflammation. In the first instance the skin becomes cold, is perhaps at first ischæmic and pale, then livid and cyanotic; bullæ may form and gangrene take place under these, or the tissue may become gangrenous *en masse* without other lesions appearing. Following traumatism death of the tissues may occur without inflammation, and the inflammatory process may only follow in the attempt of nature to throw off the dead tissue. Where occurring as a result of infection or other agencies exciting inflammation gangrene is accompanied by violent inflammatory reaction which results in the destruction of the tissues most involved. The sloughing out of a gangrenous patch is of course always associated with inflammation, and at this stage, therefore, the patch is always surrounded by a halo of inflamed tissue. Dry gangrene of the skin shows as grayish or blackish patches which at first or later have a sharply defined margin. In moist gangrene the area is purple or black; it may be covered with bullæ filled with grumous fluid; it develops in time a sharp margin around which there is more or less inflammatory areola, and finally it breaks down into a soft pultaceous mass.

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[1] Wende, *Jour. Cutan. Dis.*, 1906, p. 445.—Tsutui, *Archiv*, 1907, B. 85, p. 219.

Gangrene of the skin occurs from both external and internal causes. It may result from the action of destructive external agencies of various sorts, from violent inflammation, and from any process which obstructs the blood supply to the skin.

Crocker classifies the possible internal causes of gangrene of the skin as follows:

- I. WITHIN THE VESSEL.....
  - Embolism.
  - Thrombosis.
- II. CHANGES IN THE VESSEL WALLS..............
  - Acute arteritis.
    - *a.* Bacterial.
    - *b.* Syphilitic.
  - Calcareous degeneration, e. g., senile gangrene..............
  - Contraction of the muscular or other coats..............
    - Spasmodic, e.g., symmetrical gangrene.
    - Chronic, e. g., ergotism.
  - Trophic defects, e. g., acute decubitus....
  - Purpuric gangrene from blood extravasation.
- III. PRESSURE ON THE VESSELS FROM WITHOUT.
  - Inflammatory effusion round a vessel.
  - Tumors, etc.

Gangrene of the skin, except as the result of infection or of destructive local applications, is an extremely uncommon lesion. The most characteristic forms of it are:

Symmetrical gangrene (Raynaud's disease).

Hysterical gangrene.
Gangrene after specific infectious diseases.
Diabetic gangrene.

SYMMETRICAL GANGRENE[1]

SYNONYM: *Raynaud's disease.*

Raynaud's disease is a condition characterized by ischæmia, or congestion in terminal areas of circulation, which is accompanied by nutritional changes in the skin and in some cases by gangrene.

It is a clinical entity which is perhaps produced by various pathological conditions. The disease occurs in attacks in which the parts, most frequently the hands, are first cold and pale and then become livid and congested. The disease may not go beyond this condition, but frequently the interference with circulation is so intense that destructive changes occur. Bullæ and ulcers form, and not infrequently gangrene occurs. The gangrene may be very superficial, occurring as livid to black, dry, gangrenous patches, or there may be destructive gangrene of the entire part. The disease is usually symmetrical, and attacks most frequently the tips of the fingers, especially the pulp. Only less frequently it attacks the ears, nose, and other parts of the face, and the feet; and occasionally other parts of the body. The disease may pass off in a single attack, but it is usually recurrent and the parts become more or less permanently cyanotic. The attacks are paroxysmal in character, the paroxysms of ischæmia (syncope) or of congestion (asphyxia) lasting from a few minutes to a few hours and repeating themselves at intervals varying from a few hours apart to weeks or months, or perhaps only once in a winter. In certain attacks the parts are ischæmic, in others cyanotic. It is in connection with the cyanotic cases that destructive lesions and gangrene occur. After long duration of the disease the parts become more or less atrophic or sclerodermatous and disfigured. The nails show nutritional changes, become furrowed, abortive, and may be lost. As Beck has shown with x-rays, the nutritional changes may affect the bones.

There may be no subjective symptoms. Usually the paroxysms are accompanied by more or less pain, and there may be anæsthesia, analgesia, or thermanæsthesia.

Etiology.—The disease is most frequent in young adults. The exciting cause is in most cases exposure to cold, so that it is seen most frequently in persons whose occupations entail exposure. It is sometimes associated with mental and emotional shock, and is distinctly most frequent in neuropathic individuals. In a personal communication Bannister told me that it is not uncommon among the inmates of insane asylums. Its pathogenesis is debatable. Its paroxysmal character indicates that there is spasm of the vessels, in the case of ichæmia chiefly arterial, in the case of cyanosis chiefly venous, so that the disease is thus an angioneurosis, probably of central origin. Its paroxysmal character is strikingly analogous to that of intermittent claudication and of angina pectoris. Endarteritis has frequently been observed, but is not invariable.

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[1] Raynaud, *Thèse de Paris*, 1862, translation in publications New Sydenham Society, 1888.—Monro, "Monograph," Glasgow, 1899.—Fordyce, *Jour. Cut. and Gen.-Urin. Dis.*, 1896, p. 87.—Beck, *Amer. Jour. Med. Sciences*, 1901.—Bronson, *Jour. Cut. Dis.*, 1903, p. 456.

Diagnosis.—The combination of paroxysmal ischæmia or cyanosis occurring on the extremities and associated with bullous, ulcerative, or gangrenous lesions is pathognomonic.

Treatment.—The treatment of the bullous, gangrenous, and ulcerative lesions is along ordinary lines. During attacks hot applications should be avoided, and the parts should be rubbed with cold applications. Galvanism, after the recommendation of Raynaud, has been found useful. It is best applied by immersing the hands or feet in a solution of salt water in which the negative pole is placed, while the positive is placed over the spine or moved along the limb. The current should be used as strong as can be borne, and frequently interrupted to cause contraction of the parts. Such constitutional disturbances as exist should be remedied.*

Symmetrical Gangrene not due to Raynaud's Disease

Symmetrical gangrene not due to Raynaud's disease may, according to Crocker, occur, and he mentions the following instances:

Gangrene of the extremities, nose, and ears in a guinea pig after the insertion in the peritoneal cavity of a microbial culture in a collodion capsule (Phisalix); symmetrical gangrene of the lower extremities in suppurative peritonitis with great effusion (Vidal); a case following pulsating tumor of the brain (Treves); and one following double pneumonia (Dufour).

HYSTERICAL GANGRENE[1]

SYNONYMS: *Neurotic Gangrene, Spontaneous Gangrene, Erythema Gangrænosum.*

As has been seen under feigned eruptions, the occurrence of gangrene in hysterical subjects is usually the result of feigning. The occurrence of such lesions in an hysterical individual always suggests the probability of self-infliction, and many authors class all cases of hysterical gangrene as feigned eruptions, assuming that in a few apparently spontaneous cases the trick was not detected. There seems good reason to believe, however, that in excessively rare cases spontaneous bullous and gangrenous lesions may occur in hysterical subjects. Such cases have been recorded by Doutrelepont, Duhring, Schwimmer, Kaposi, and others, and a very convincing case of bullous dermatitis in an hysterical subject in which malingering seems to be definitely excluded has been recorded by C. J. White.

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[1] **White, C. J.**, *Jour. Cut. Dis.*, vol. xxi, 1903, p. 415.—Doutrelepont, *Archiv f. Derm. u. Syph.*, vol. xiii, 1886 and 1890.—Joseph, *Archiv f. Derm. u. Syph.*, vol. xxi, 1895.—**Duhring**, "International Atlas of Rare Skin Diseases," Plate XLVIII.

Most of the cases are in women, but some are in men. The patients are extremely neuropathic, usually with characteristic stigmata of hysteria. In most cases the condition begins with a slight injury, after which the lesions appear. In some cases they are at first bullous and then gangrenous, in other cases gangrenous from the start. The distribution is quite irregular and the mucous membrane of the mouth may be involved.

Zoster Atypicus Gangrænosus et Hystericus

Under this term Kaposi has described eleven cases characterized by vesicular and gangrenous lesions which he was sure were not due to malingering. They occurred in hysterical individuals, but not all of them in women. Their nosological position is uncertain, and if they were not feigned eruptions there seems no good reason to separate them from the other cases of hysterical gangrene.

ECTHYMA GANGRÆNOSUM[1]

SYNONYMS: *Varicella grangrænosa* (Hutchinson), *Pemphigus gangrænosus* (Whitely Stokes), *Rupia escharotica* (Fagge), *Ecthyma térébrant* (Fr.), *Ecthyma Gangrænosum*, *Dermatitis Gangrænosa Infantum.*

Dermatitis gangrænosa infantum is applied to a rare eruption of gangrenous lesions which supervene upon varicella and other pustular eruptions in children, and usually occur spontaneously.

Hutchinson, whose description of various cases under the title varicella gangrænosa called attention to this condition, first thought that it followed only varicella and vaccinia, but it is now known to occur after other pustular eruptions and spontaneously. It is probable that the gangrenous eruptions which occur at times in all of the exanthemata and in purpura and erythema nodosum, usually in children but occasionally also in adults, are illustrations of the same condition. The French term ecthyma térébrant, and especially the German term, ecthyma gangrænosum, well describe the disease, and as they do not predicate anything as to age, are the preferable terms.

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[1] Crocker, "Diseases of the Skin," p. 535; *London Med.-Chirurg. Soc. Trans.*, 1887.—Hutchinson, "Clinical Lectures on Rare Diseases of the Skin," p. 235; *London Med.-Chirurg. Soc. Trans.*, 1882, p. 1.—Baudouin et Wickham, *Annales*, December, 1888.—Elliot, *Med. Record*, May 16, 1891, p. 862.—Hitschmann, Fritz, und Kreibich, *Wien. klin. Wochenschr.*, 1897, No. 50; *Archiv*, 1899, vol. i.

Symptomatology.—The condition most frequently supervenes upon the eruption of varicella, and its course with varicella may be regarded as the typical one. The pustule, instead of drying up in the usual way, ulcerates with the formation of a central grayish or blackish crust surrounded by a pustular border, the whole upon an inflammatory base. The gangrenous ulceration spreads peripherally, as well as in depth, forming ultimately a gangrenous slough of variable size up to an inch or more in diameter. A line of demarcation finally forms, the slough is thrown off, and a round or oval conical ulcer is left of a depth corresponding to its extent. The individual ulcers are thus very much like gangrenous ulcers which are sometimes seen at the points of vaccination. Adjacent ulcers tend to coalesce, and, as the lesions may be very abundant, large irregular ulcers may be formed, or in extensive cases the surface may be riddled with ulceration. Where the disease develops spontaneously small pustules appear which spread and ulcerate with the formation of lesions similar to those described above, but usually smaller. Where the condition complicates varicella the distribution of the lesions is the same as that of the lesions of varicella. In other cases the eruption is apt to be most abundant about the buttocks, the lower part of the back, and the legs and thighs where they are covered by the napkin. Over the rest of the body the lesions are fewer. In a few rare cases single large gangrenous ulcers have been observed in infants, occurring spontaneously, and also after specific infectious diseases. The patches have begun as vesicles, pustules, or bullæ, under which gangrenous spreading ulceration has occurred.

The disease may appear as one crop of gangrenous ulcers, but usually the first eruption is followed irregularly by other similar but smaller lesions. It runs a course of two to four weeks. After the gangrenous sloughs separate, the ulcers heal quickly, and even when the illness has been grave, recovery may be rapid.

The eruption presents great variations in intensity. In the mildest cases the ulceration is very superficial, very like that of the ulceration of typical vaccination. In the ordinary type of cases there is considerable destruction of connective tissue, and in extreme cases the ulceration may be three quarters of an inch in depth. The lesions may at times begin as bullæ, and in other cases they have been hæmorrhagic.

The disease is accompanied by constitutional symptoms of a septic character corresponding in intensity with the gravity of the eruption. The temperature may reach 103° to 105° F., and the complication of general sepsis occur. In the severest cases death in eleven to twelve days has taken place.

Etiology.—All of the cases complicating varicella have been seen in early childhood, the greatest number under one year of age and a large majority of cases under three years. It is much more common in girls. It has usually occurred in debilitated children and tuberculosis has been present in many cases, while in a few cases it has occurred in apparently healthy children. Although the disease occurs characteristically in children, a similar multiple gangrene has been observed in adults after scarlatina, measles (Hutchinson), suppuration in the vagina (Crocker), malaria (Osler), typhoid fever (Hahl), and many cases with enteric fever and smallpox. Other cases in adults have been traced to various infections—e. g., with a hypodermic syringe (Waelsch) or the wound made by a meat hook (Hartzell). This case of Hartzell's suggests the similarity of the condition to the cases of acute pemphigus following animal poisons, such as have been reported by Pernet.

Of its actual causation we are in ignorance. It is probably in most cases due to local infection in a cachectic person, but it is possibly at times a manifestation of general infection. Various bacteria have been found in the lesions—the bacillus pyocyaneus (Ehlers and others), the bacillus ramosus (Veillon and Halle), the streptococcus pyogenes (Baudouin and Wickham)—but the pathogenic character of none of these is established.

Diagnosis.—The appearance of gangrenous ulcers, more or less generalized and bilateral, in children and usually as a complication of varicella or other exanthems, with constitutional symptoms corresponding in intensity with the extent of the eruption, is a characteristic symptom complex. From congenital syphilis it is readily differentiated by its acute onset, by the absence of other lesions of syphilis, and by the absence of the stigmata of congenital syphilis.

Treatment.—The local treatment is the use of antiseptic baths and dressings, in the form of wet dressings preferably, or ointments. The internal treatment is directed to supporting the patient as fully as possible.

Diabetic Gangrene

Diabetic gangrene is gangrene which occurs in profound conditions of diabetes either spontaneously or as a result of traumatism.

Compared with the entire number of cases of diabetes, gangrene is relatively very uncommon, but it is not itself an excessively rare condition. It may occur as dry gangrene, but is more frequently moist.

In diabetic gangrene the involved areas may become gangrenous directly, first becoming livid, and then dry, grayish, or black. In other cases there may be first the appearance of vesicles or blebs under which the gangrene occurs.

Sometimes there occur small, dry, grayish, or black gangrenous patches which hardly involve more than the corium. After several days they separate, leaving a granulating surface which heals slowly. Usually the gangrene extends well into the subcutaneous tissues, and it is likely to occur in large patches. Sometimes it may involve all of the tissues, destroying one or more of the toes, or part or all of the foot, or in the gravest cases an entire limb. It may begin spontaneously, but more frequently it follows some slight traumatism. It occurs most frequently on the feet and legs, but at times on the upper extremities. Gangrene is a manifestation of grave conditions of diabetes, and is seen nearly always in patients in later life. In twenty-three of twenty-four cases Wallace found atheromatous arteries and an average age of sixty-three years.

In cases following wounds, infection in tissues whose resistance is damaged by the presence of sugar in the blood is usually invoked to explain the gangrene. The spontaneous forms of the disease, however, do not pursue the course of external infection, but rather suggest a condition in which the blood supply of the part is cut off from within, either by embolism or, more frequently, by endarteritis.

Syphilitic Gangrene

In rare instances gangrene is produced by syphilitic endarteritis. It occurs as a dry gangrene of an extremity, usually involving fingers or toes, and is accompanied by severe pain. It occurs in old syphilitics, and is caused by gummatous endarteritis obstructing the blood flow to the involved part. Vigorous mixed specific treatment is indicated.

Phagedena Tropica[1]

Synonyms: *Tropical phagedenic ulcer, Aden ulcers, Malabar ulcers, etc.*

Under this name are described forms of phagedenic and gangrenous ulceration which are observed as the result of inoculation in cachectic individuals in tropical and subtropical climates.

It may occur as chronic phagedenic ulcers developing with acute inflammatory onset and spreading over the surface, or as deep gangrene involving all the tissues, even including the bones. Unless quickly fatal it pursues an indolent chronic course, perhaps extending over a year or two, but ultimately healing.

The disease has been observed in the countries along the Asiatic and

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[1] Crocker, p. 544.—Hirsch, "Phagedenic Tropical Ulcers," vol. iii, p. 690, *Syd. Soc. Edit.*—Parke, "The Ulcer of the Emin Pasha Relief Expedition," *Lancet*, December 5, 1891.

African shores of the Indian Ocean, in Central Africa, Egypt, Algiers, the West Indies, and Central America. It occurs in cachectic individuals who have been reduced in health by the privations and the unsanitary surroundings of a tropical climate and by malaria and other tropical diseases. According to Boinet it is due to a specific aërobic bacillus, and Le Dantec identifies it with hospital gangrene.

IV.—DERMATITIS MEDICAMENTOSA[1]

Synonyms: *Drug eruptions.*

Introductory.—The term dermatitis medicamentosa is applied t eruptions arising from the action of drugs. It is frequently limited t eruptions produced by the internal action of drugs, eruptions produce by their external action being included under dermatitis venenata. Bu it is not possible to draw a hard and fast distinction of this sort, and i is more logical to consider together all of the eruptions produced by an given drug regardless of whether its action is internal or external.

The eruptions produced by external contact with drugs can be dis cussed very briefly. They are in nearly all instances forms of simpl dermatitis, quite like those produced by other external irritants. Ver rarely there is a peculiar eruption produced by the local action of a dru as folliculitis produced by tar or by the constant contact of oils, but is hard to find further illustrations of this sort. Inflammatory process excited by the external application of drugs vary in degree according the quality of the irritant, the duration and concentration of its appl cation, and according to the vulnerability of the skin. Thus, with t lightest application of croton oil one can produce a superficial dry d matitis; by repeating the applications one can produce all grades reaction through vesicles, bullæ, and pustules to gangrene.

The eruptions which the internal action of drugs may excite run t entire gamut of cutaneous lesions. They may be macular, papular, ves ular, bullous, urticarial, pustular, ulcerative, nodular, and gangreno The eruptions produced by any drug vary in different cases; on the oth hand, different drugs may produce the same eruption in different indivi uals. A complete consideration of drug eruptions therefore would tou upon nearly the entire domain of dermatology, and it is not possible describe in detail all of the eruptions produced by different drugs. Mo over, it is not necessary, for while drug eruptions have certain cha acteristic features, they, in their various forms, closely simulate defin dermatoses; indeed, in many cases they may be regarded as definite dermatoses, the only peculiarity being the cause. This simulation of dermatoses by drug eruptions throws an interesting side light upon etiology; for, if a definite dermatosis, like a papular erythema or an urticaria, can be produced by a drug, it is highly probable that other instances of these same dermatoses are produced by other poisons and are in some, if not in all, cases toxic dermatoses.

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[1] Morrow, "Drug Eruptions," Wm. Wood & Co., New York, 1887.—Ehrman *Mracek's Handbuch.*—Van Harlingen, *Amer. Archiv. of Derm.*, vol. vi, p. 337. Brooke, *Brit. Jour. Derm.*, 1890, p. 313.—Stowers, *Brit. Jour. Derm.*, 1898, p. 289. Pernet, *Brit. Med. Jour.*, May 16, 1903.

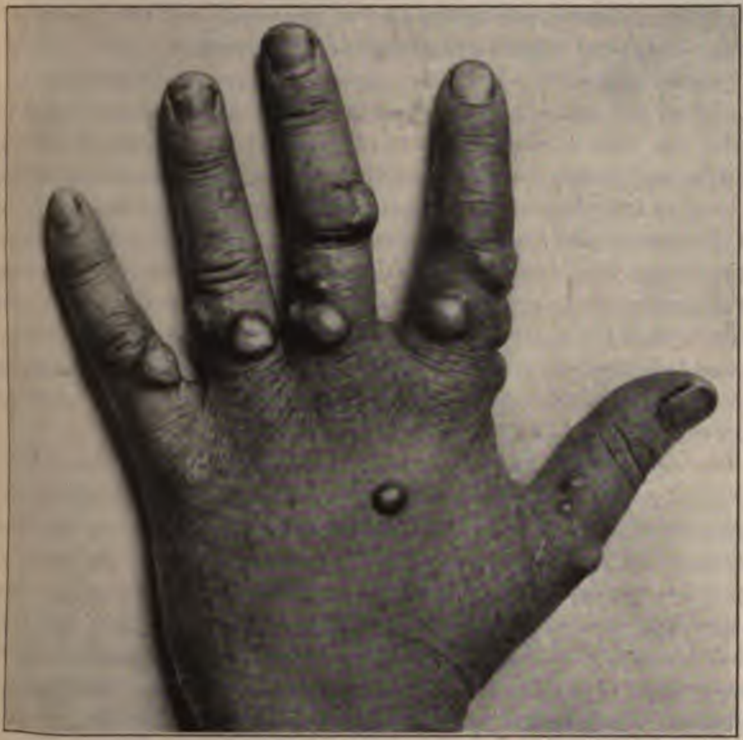

FIG. 87.—BULLOUS DERMATITIS FROM IODINE OINTMENT.
(Grover W. Wende's collection.)

Speaking broadly, the eruptions produced by the internal action of drugs resemble those dermatoses, presumably of toxic origin, which are the result of vasomotor or trophic disturbances. Most frequently, drug eruptions simulate the vasomotor disturbances which are characterized by various degrees of inflammatory reaction, like the hyperæmic erythemas, erythema multiforme, and urticaria. Occasionally they simulate the more violent trophic disturbances which are characterized by vesicles, bullæ, ulceration, or even gangrene. They are not ordinarily forms of simple dermatitis such as can be produced by external irritation, and the term dermatitis as applied to them is misleading.

The drug eruptions are usually bilateral and more or less symmetrical. They may vary from a single lesion or two up to a universal eruption. Their areas of predilection vary with different eruptions, but they appear most frequently and abundantly upon the face, the hands and wrists, the neck and upper part of the chest, and the legs below the knees. They may occur on the mucous membranes.

Sudden appearance is a characteristic feature of drug eruptions, especially of the acute inflammatory eruptions, and they are apt to be a livelier red than is usual for the eruptions which they simulate. Their duration and course is dependent upon the action of the exciting drug. So long as it is continued they are apt to become more intense in their manifestations, and they usually disappear quickly upon the elimination of the drug. This does not apply to the eruptions of bromine and iodine. These usually occur only from the cumulative effect of the drug in the system and are not seen, especially in their graver forms, at the beginning of administration. The eruptions from iodine and bromine may indeed begin after the drugs have been stopped, and they are correspondingly slow to disappear.

The acute inflammatory eruptions produced by drugs are characterized by a considerable degree of itching, burning, or tingling. The indolent types of drug eruptions are usually without much disturbance of sensation. Drug eruptions of slight extent are, as a rule, not accompanied by constitutional symptoms, but in eruptions of wide extent, as, for example, generalized scarlatiniform eruptions, there may be a sharp systemic reaction with the usual accompaniments of a moderate febrile disturbance. If the administration of a drug is persisted in after it has produced violent manifestations in the skin, the constitutional symptoms may become grave, and in excessively rare cases there have been fatal results.

Etiology and Pathology.—The factors which predispose to the production of dermatitis from the external action of drugs are the same as those which predispose to the production of dermatitis from other forms of external irritation, and need no discussion here.

The number of drugs which at times may produce eruptions of internal origin is almost as great as the number of drugs which are used. At the same time most drugs very rarely produce eruptions, and there are only a few drugs which, like iodine and bromine, often produce cutaneous manifestations. Of these iodine is most commonly associated with eruptions. With it some evidence in the skin is so frequently an accompaniment of its administration as to be one of its usual effects.

Drug eruptions are more common in women and children. They are also more common in neuropathic individuals and in individuals in a lowered state of health. Renal and cardiac diseases are the most potent of pathological conditions in their production, but the chief factor in their production is idiosyncrasy. This is of variable degree. In some instances it amounts to a sensitiveness almost incredible, infinitesimal quantities of the peculiar drug to which the individual is susceptible producing violent and extensive cutaneous reaction.[1] The idiosyncrasy in a given individual may in some cases disappear with persistence in the administration of a drug, but as a rule it becomes more sensitive with repeated intoxications.

From the analogy of eruptions due to other forms of intoxication there seems every reason to believe that in most instances drug eruptions result from the action of toxic substances upon the vasomotor centers controlling the circulation in the skin or upon the trophic centers of the skin. These toxic substances in most instances are probably the drugs themselves; perhaps at times they are materials elaborated in the body from the action of the drugs. In certain conditions, especially with the bromides and the iodides, the skin lesions seem to be produced in large part by the irritation arising from the elimination of the drugs through the skin.

Diagnosis.—Drug eruptions are apt to mislead one unless he has in mind the possibility of their existence in a given case and investigates the immediate previous history of the patient as regards the medicines he has taken. They are distinguishable from the diseases which they simulate by their sudden appearance, their atypical course, and the absence of characteristic symptoms aside from those in the skin.

Treatment.—The local treatment of drug eruptions is symptomatic. In the general management the first thing is the withdrawal of the suspected drug. With drugs like bromine and iodine, whose action persists, eliminating treatment is indicated—the drinking of abundance of water, perhaps other diuretics, and the use of aperients and cathartics.

The eruptions from various drugs are indicated below, but it should not be forgotten that these eruptions show great variations and that, barring the peculiar iodide and bromide, silver and arsenic lesions, there is hardly any eruption produced by one drug that may not be produced by another. The following summary is taken almost entirely from Stelwagon:

Alopecia.—Thallium acetate, boric acid.

Acne.—Bromine, iodine, internally; tar, oils, and fats, externally.

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[1] Stelwagon, *Jour. Cut. and Gen.-Urin. Dis.*, 1902, p. 13.

Bullous.—Aconite, anacardium, antipyrine, boric acid, chloral, bromine, quinine compounds, copaiba and cubebs, iodine compounds, iodoform, mercury, opium (?), phosphoric acid, and salicylates.

Carbuncular (Anthracoid).—Arsenic, chloral, iodine and bromine compounds, and opium.

Cyanotic.—Acetanilide, potassium chlorate.

Dermatitis.—All irritant drugs.

Erysipelatous.—Arsenic, belladonna, conium, digitalis, ipecac, quinine, and stramonium.

Erythematous.—Acetanilide, antipyrine, arsenic, alcohol, antitoxin, belladonna, benzoic acid, boric acid, bromine compounds, capsicum, carbolic acid, chinoline, chloral, chloralamide, cantharides, chloroform, castor oil, conium, copaiba, cubebs, dulcamara, exalgin, iodine compounds, iodoform guaiacum, gurjun oil, hydrocyanic acid, hyoscyamus, lead acetate, mercury, opium, pilocarpine, piper mythysticum, phenacetin, phosphoric acid, potassium chlorate, quinine, salicylates, sodium benzoate, santonin, sodium borate, stramonium, sulphonal, tannic acid, tar, oil of turpentine, tuberculin, veratrum viride, and eucalyptus oil.

Erythemato-papular.—Acetanilide, antipyrine, benzoic acid, copaiba, digitalis, gurjun oil, iodide compounds, iodoform, phenacetin, silver nitrate, and potassium chlorate.

Epitheliomatous.—Arsenic (secondarily to keratoses).

Furuncular.—Antipyrine, arsenic, bromine compounds, calx sulphurata, chloral, condurango, ergot, mercury, and opiates.

Gangrenous.—Arsenic, belladonna, ergot, iodine compounds, quinine, salicylates.

Herpetic.—Arsenic, belladonna, iodine compounds, mercury, and salicylates.

Keratotic.—Arsenic.

Morbilliform.—Antipyrine, antitoxin, belladonna, copaiba and cubebs, boric acid, opium, sodium borate, sulphonal, tar, turpentine, and tuberculin.

Nodular.—Iodine and bromine compounds.

Papillomatous.—Iodine and bromine compounds.

Papular.—Arsenic, boric acid, bromine compounds, cantharides, chloral, conium, copaiba and cubebs, cubebs, digitalis, iodine compounds, jaborandi, oil of turpentine, mercury, terebene, and opium.

Papulovesicular.—Capsicum.

Pigmentary.—Arsenic and silver nitrate.

Pruritus (without Eruption).—Opium, chloral, copaiba, strychnine.

Purpuric (including Petechial).—Antipyrine, antitoxin, arsenic, benzoic acid, calx sulphurata, chloral, chloroform, copaiba and cubebs, ergot, hyoscyamus, iodoform, iodine compounds, lead acetate, mercury, phosphoric acid, potassium chlorate, oil of sandalwood, quinine, salicylates, stramonium, and sulphonal.

Polymorphus (resembling Erythema Multiforme).—Antipyrine, antitoxin, sodium benzoate, copaiba and cubebs, iodine compounds, iodoform, boric acid, chloral, coal-tar derivatives, opium, potassium chlorate.

Psoriasiform.—Sodium borate and tuberculin.

Pustular.—Aconite, antipyrine, arsenic, bromine compounds, calx sulphurata, condurango, antimony, hyoscyamus, iodine compounds, ergot, mercury, nitric acid, cod-liver oil, opium, oil of turpentine, salicylates, and veratrum viride.

Papulo-pustular.—Bromine and iodine compounds.

Scarlatiniform.—Antipyrine, antitoxin, belladonna, chloral, copaiba and cubebs, copaiba, digitalis, hyoscyamus, mercury, nux vomica, opiates, oil of turpentine, pilocarpine, rhubarb, quinine, strychnine, sulphonal, salicylates, stramonium, tuberculin, and viburnum prunifolium.

Ulcerative.—Arsenic (secondarily to keratoses), bromine compounds, chloral, iodine compounds, and mercury.

Urticarial.—Alcohol, antimony, anacardium, antipyrine, antitoxin, arsenic, bromine compounds, benzoic acid, chloral, copaiba, cubebs, digitalis, dulcamara, hydrocyanic acid, guarana, hyoscyamus, iodine compounds, opium, mercury, pilocarpine, phenacetin, pimpinella, quinine, salicylates, salol, santonin, oil of turpentine, sodium benzoate, tannin, tar, and valerian.

Vesiculo-pustular.—Antimony, antipyrine.

Vesicular.—Aconite, anacardium, antimony, antipyrine, arsenic, bromine compounds, cannabis indica, calx sulphurata, chloral, copaiba, cubebs, cod-liver oil, ergot, iodine compounds, iodoform, nux vomica, oil of turpentine, opium, quinine, salicylates, and sodium santonate.

The eruptions produced by a few drugs deserve further consideration, either on account of their practical importance or peculiar features.

Antitoxin.—Macular and papular erythemas and urticaria, either separately or together, develop very frequently after the use of antitoxin. The eruption may appear in a few hours after the administration or not for several days, and persists for from a few days to one or two weeks.

Acetanilide.—Macular and maculo-papular erythemas and peculiar slate-colored cyanosis.

Antipyrine.—Various forms of macular and papular erythemas; urticaria; in some cases vesicular, bullous, and purpuric lesions. May involve mouth.

Copaiba, Cubebs, and Sandal Oil.—Copaiba frequently, and cubebs occasionally, produce erythemas. They may be generalized, scarlatiniform, or morbilliform eruptions, but most frequently they occur as not very abundant eruptions of large macules or maculo-papules up to the size of a finger nail, of a bright-red color, of irregular shape, and most abundant upon the face. Sandal oil has been observed to produce a purpuric eruption, but it is a rare occurrence.

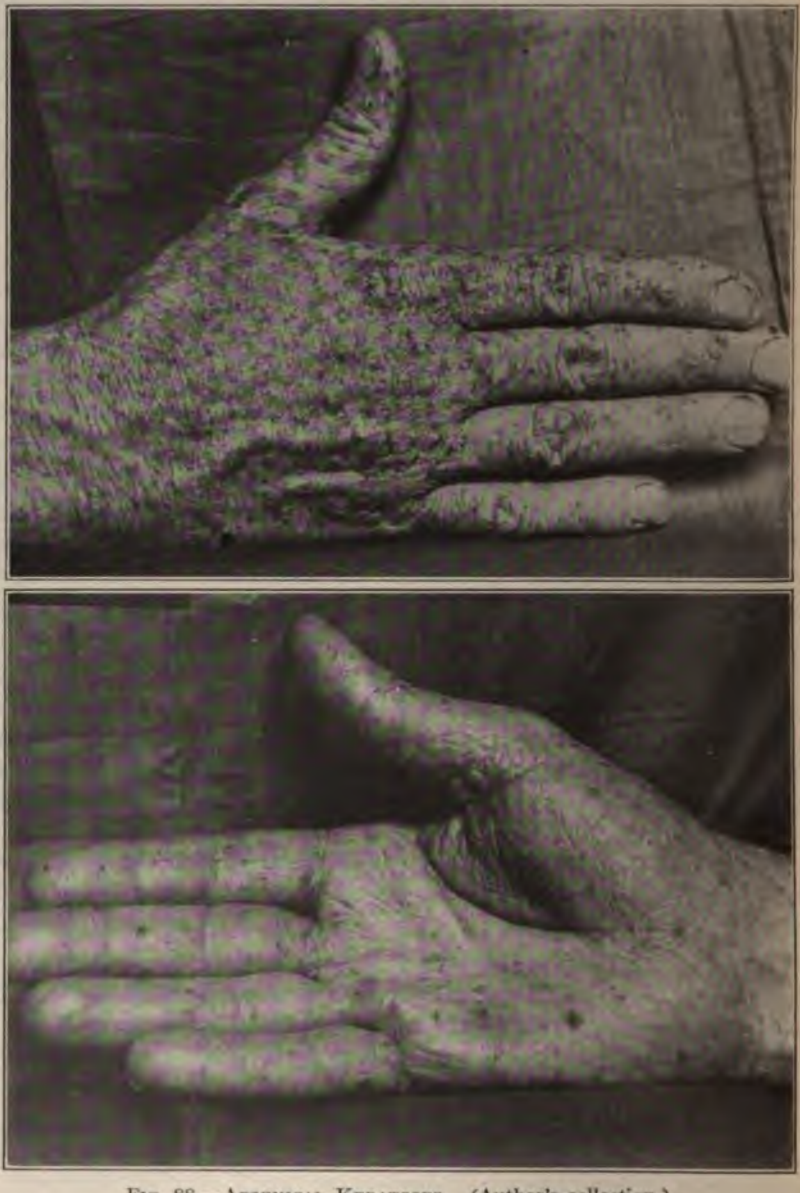

FIG. 88.—ARSENICAL KERATOSES. (Author's collection.)

Iodoform.[1]—Iodoform is a not uncommon cause of various degrees of dermatitis. It may be a dry erythematous dermatitis, but more frequently it is a rather acute papulo-vesicular and occasionally bullous dermatitis. It may spread widely beyond the point of application of the drug. I have seen a universal acute erythemato-vesicular dermatitis produced from once dressing an incised bubo with iodoform. The absorption of iodoform may produce erythematous, urticarial, and purpuric eruptions.

Orthoform. — Dermatitis similar to that of iodoform. Its frequent application may cause gangrene at the point of application.

Chrysarobin.—Its local application produces a brownish-red erythematous dermatitis which spreads beyond the site of application. On the face it may cause great swelling and conjunctivitis.

Arsenic.[2] — The various eruptions produced by most other drugs are in very rare cases produced by arsenic. In addition it has been observed to produce ulcerative, œdematous, and gangrenous lesions, especially about the scrotum. As a sequel of neuritis, which it occasionally produces, zoster may occur. Its long-continued administration may produce pigmentation especially over the trunk, which usually ultimately disappears. In occasional rare cases its long-continued administration in diseases like psoriasis may be followed by keratoses of the hands and feet. In a case of this sort under my observation, the condition was

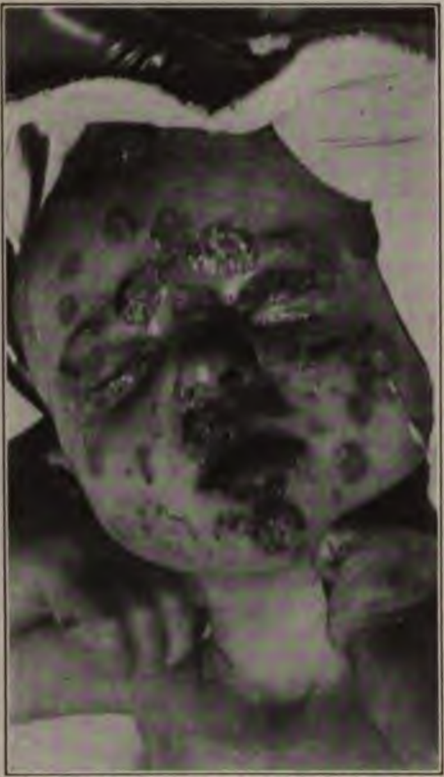

FIG. 89.—IODERMIA, FROM MOTHER'S MILK; FATAL CASE. (Author's collection.)

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[1] Fox, Colcott, *Brit. Jour. Derm.*, 1890, p. 327.—Taylor, *N. Y. Med. Jour.*, October 1, 1887.—Cutler, *Boston Med. and Surg. Jour.*, 1886.

[2] Brooke and Roberts, *Brit. Jour. Derm.*, vol. xiii, 1901, p. 121, "The Study of a Large Number of Cases of Skin Lesions Occurring in an Epidemic of Arsenical Beer Poisoning."—Hamburger, *Johns Hopkins Bulletin*, April, 1900.

well marked not only upon the palmar, but also upon the dorsal su of the hands. The conditior the dorsal surfaces with nur hyperkeratoses upon hyp bases presented a picture n like that of chronic x-ray titis of the hands. Epithel may develop in these hyp toses.[1]

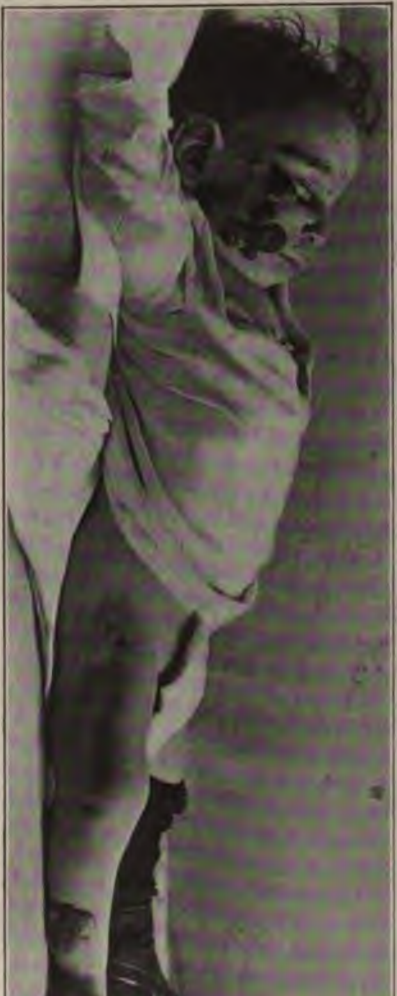

FIG. 90.—BROMIDE ERUPTION. (Pollitzer's collection.)

Silver.—The long-contin ministration of silver may lowed by a permanent, bluis pigmentation of the skin, es of the exposed parts (argyri pronounced cases the color changed to a dark slate color pigmentation is peculiar and to a permanent deposit of si the skin. From its greater sity upon exposed parts it likely that its explanatio in the decomposition by li the silver salts circulating blood. It is thus a sort of ing from within. Gilchri found silver in the corium gyria.

Bromine.[2]—Eruptions bromine, usually administe the form of bromides, ar common. Its usual eruptio the form of an acne which hardly distinguishable from dinary acne. The productio few pustular acne lesions usual occurrence upon the istration of bromides to p who have a tendency to acne characteristic lesions of bromide acne are large, indolent, purplish-red pustules. These show a tendency to occur in groups and to coalesce into more or less rounded sharply elevated lesions of the size of a coin or larger, whose surfaces are studded with the openings of follicles distended with pus. In exaggerated cases these lesions develop into large, hypertrophic tumors with an irregularly globular surface which is pitted by the conical depressions corresponding to the openings of the follicles. They are of dark-brownish purple, sometimes two or three inches in diameter, and may or may not discharge pus from the follicles. Except for their raspberrylike surface they suggest sarcomata. The exaggerated bromide lesions are usually few, and occur most frequently about the legs. They are practically painless. The acne lesions occur chiefly on the face and shoulders, in the distribution of ordinary acne. Lesions on the legs from bromides simulating erythema nodosum have been described by Voisin and Veiel. In addition to these characteristic eruptions, in very rare cases the bromides have excited the commoner vasomotor drug eruptions.

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[1] Hartzell, *Amer. Jour. Med. Sci.*, vol. cxviii, 1897, p. 205.—Darier, vol. iii, 1902, p. 1126.

[2] Van Harlingen, *Trans. Amer. Derm. Assn.*, 1880; *Arch. of Derm.*, 1880.—Jackson, *Jour. Cut. and Gen.-Urin. Dis.*, 1895, p. 462.—Elliot, *Tran Derm. Assn.*, 1895.—Engman, *Trans. Amer. Derm. Assn.*, 1906.

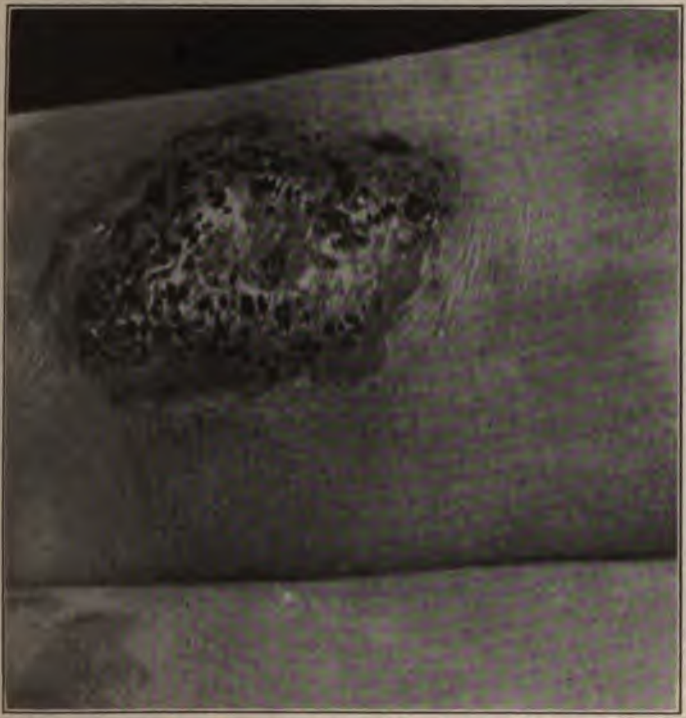

FIG. 91.—LARGE SINGLE LESION OF BROMIDE ERUPTION ON LEG. A large dark red frambesiform tumor, with greatly dilated follicles; no suppuration. (Author's collection.)

The lesions may not appear until the bromides have been taken for some time. They appear in some cases after the discontinuance of the bromides. They are sluggish in their course, and the large hypertrophic lesions may persist for several months.

The hypertrophic bromide eruptions are most frequent in children, and may occur in suckling children from the use of bromides by the mother. Defective renal elimination is a factor in their production, but idiosyncrasy is probably more important. They are usually seen after prolonged use of bromides in large doses.

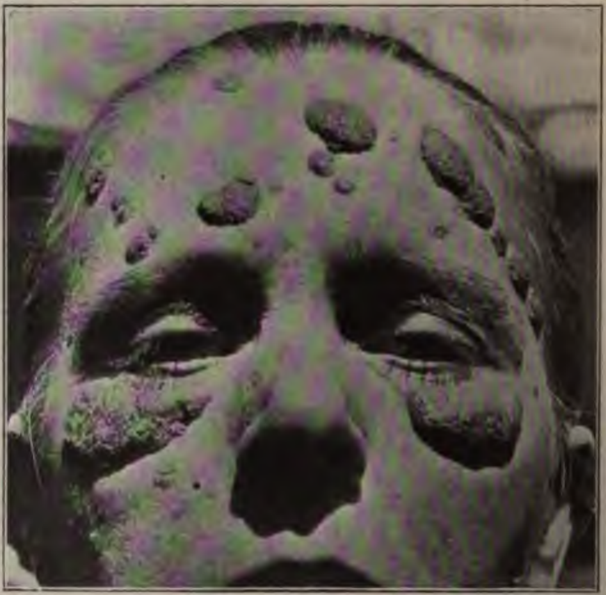

Fig. 92.—Frambesiform Lesions from Iodine (KI). (C. J. White's collection.)

Pasini[1] believes that bromine eruptions are caused by the setting free of bromine from its salts by some change in the blood, and he suspects, on the basis of researches made by Fèrè, Voisin, and others, that a diminished chloride content is the cause of this reaction.

Bromide acne is benefited by the use of arsenic and may be almost prevented, where the use of the drug is necessary, as in epileptics, by its administration. The local treatment is that of indurated acne. In addition the bowels should be kept open, free catharsis should occasionally be produced, and elimination by the kidneys provided for as far as possible.

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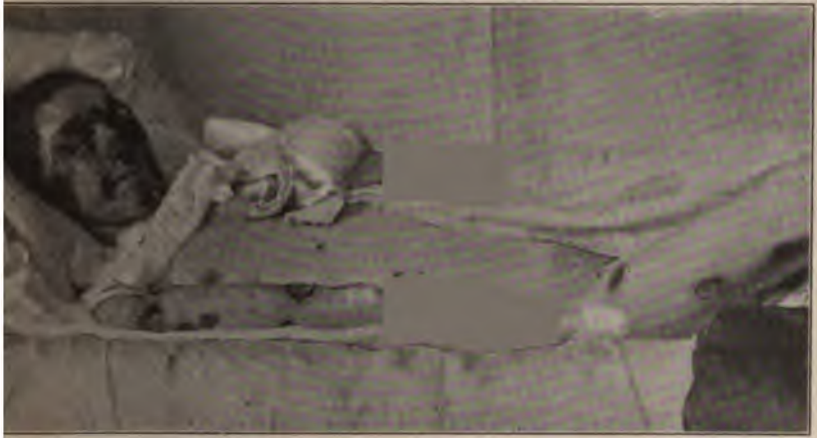
[1] Pasini, *Brit. Jour. Derm.*, 1907, p. 42.

Iodine.[1]—Iodine and its compounds are more constantly associated with eruptions than any other drugs. Occasionally erythematous, urticarial, vesicular, and papular lesions may be produced by it, but these are decidedly uncommon. Its most frequent eruption is an acne about the face and shoulders, which differs from the bromide acne in that the lesions are smaller and are not of the same indolent character and purplish color. As a rule they are discrete, but they may be confluent and produce the conglomerate, frambesiform lesions which are more characteristic of the bromides. At other times the lesions are condylomatous. In other cases there may be bullæ along with the other lesions, and in some cases the eruption is chiefly bullous. The bullæ are usually situated upon an erysipelatous base, and they may be followed by ulceration and even gangrene. In other cases nodular sarcomalike tumors form, which may ulcerate. In very rare cases purpura occurs, and erythema odosum has been observed.

Iodine acne has the distribution of ordinary acne. The grave iodide ruptions occur by predilection upon the face and extremities. Their urse is indolent and similar to that of the grave bromide eruptions. he purpuric and bullous and severe nodular eruptions may be accom-anied by severe constitutional disturbances and have been fatal in ex-eme cases. The etiology is similar to that of the bromide eruptions,



. 93.—Bullous and Ulcerative Lesions from Iodine (KI), resulting in Death. (Author's collection.)

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[1] Montgomery, D. W., *Trans. Med. Soc. of Cal.*, 1900 (with Bibliography).—osenthal, *Archiv f.*, vol. lxxii, 1901, p. 3.—Walker, *Lancet*, May 12, 1892.—ordyce, *Jour. Cut. and Gen.-Urin. Dis.*, 1895, p. 496.—Neumann (Fatal Case), *rchiv*, vol. xlvii, 1899, p. 324.

and renal and cardiac diseases are distinctly associated with them. Th treatment is similar to that of bromide eruption.

Mercury.—Mercury and its salts are irritating, and, as is well known, produce dermatitis. Very rarely does the absorption of mercury produce eruptions, although the common drug eruptions have been observed from its use. Tomascewski [1] has cited cases where mercury produced diffuse erythematous eruptions and macular erythemas (both measles-like and scarlatina-like), urticaria, purpura, and bullous (pemphigoid) eruptions—all of the toxic angioneurotic types of eruption. It is important to know, however, that mercury practically never produces an eruption which should cause any confusion with a syphilide, except possibly a macular syphilide.

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[1] *Zeitschrift für klin. Med.*, vol. ii, Nos. 5 and 6.

SECTION V

DRY SCALY INFLAMMATORY DERMATOSES

In this class are included a number of dermatoses which occur uniformly as dry, scaly eruptions. The group is a clinical one, including affections which are of widely different features but at the same time show many common characteristics. Between some of them there are transitional forms. They are all inflammatory dermatoses, but they are not forms of simple dermatitis; they are not forms of dermatitis which can in any way be excited by external irritants. Nearly all of them are definite clinical entities. In hardly any of them do we know the essential cause, but most of them are so definite in their manifestations that there is little doubt that they are distinct pathological as well as clinical entities. Most of them are stable dermatoses; that is, dermatoses presenting constantly one type of lesions which show little tendency to essential variations. A dry, inflammatory papule or spot, with more or less tendency to scaling when it becomes grouped with other papules, is the elementary lesion in practically all of these affections.

I.—LUPUS ERYTHEMATOSUS[1]

SYNONYMS: *Seborrhœa congestiva* (Hebra), *Lupus erythematodes*, *Lupus superficialis* (Parkes and Thompson), *Lupus sebaceus*, *Erythème centrifuge* (Biett), *Ulerythema centrifugum* (Unna).

Lupus erythematosus is a disease of the skin usually pursuing a chronic course, and characterized by circumscribed, pink to red, more or less scaly patches which, when they undergo involution, are followed by atrophic scars.

Lupus erythematosus has been described by different authors under the various names given above. The designation now universally applied to the affection was given it by Cazenave in 1850.

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[1] Jamieson, *Brit. Jour. Derm.*, 1893, p. 115.—Rona, *Archiv*, 1901, vol. lvi, p. 381.—Sequeira, *Brit. Jour. Derm.*, 1903, p. 171.—Fordyce, *Jour. Cut. Dis.*, 1896, p. 89.—Hallopeau, *La sémaine med.*, 1898, vol. xviii, p. 225.—Fordyce and Holder, *Med. Record*, New York, July 14, 1900.—Crocker, *Brit. Jour. Derm.*, 1898, p. 375.—Warde, *Brit. Jour. Derm.*, 1902, pp. 332 and 380, and 1903, p. 161.—Galloway and MacLeod, *Brit. Jour. Derm.*, 1903, p. 81.—Polland, *Dermatolog. Zeitschr.*, 1894, p. 482.—Breda, *Berl. klin. Wochenschr.*, 1902, No. 30.—Gunsett, *Münch. med. Wochenschr.*, 1903, No. 9.—Voirol, *Deutsche Mediz. Ztg.*, 1903.—Poór, *Dermatolog. Zeitschr.*, 1901, vol. viii.—Anthony, *Jour. A. M. A.*, January 10, 1903.

The frequency with which the appellation lupus has been applied to it is evidence of its clinical resemblance to lupus vulgaris. This is dependent chiefly upon the fact that it usually occurs in circumscribed patches about the face. The name, as suggesting an essential relationship to lupus vulgaris, is unfortunate. Except in distribution and in its broadest features it does not, as a rule, suggest lupus vulgaris, and it is not a form of tuberculosis of the skin.

It is not a common disease, but in America is probably quite as frequent as lupus vulgaris.

Symptomatology.—The beginning lesion of lupus erythematosu is a slightly elevated flat papule of pinhead to tackhead size, and o a bright-red color which does not entirely disappear under pressure The center of these lesions shows a thin scale or slight atrophy. Thei course is very chronic, but gradually they enlarge by peripheral extension and thus form sharply circumscribed, discoid or rounded, slightl elevated patches, which are more or less infiltrated, of pinkish to bright red color, slightly scaly, and characteristically studded with gapin follicles which are plugged with dry sebum and epithelial *débris*. The infiltration in the patches varies considerably. It may be hardly appreciable and the patches may be without elevation; usually it is firm an the patches, at least at the borders, are definitely elevated above the surrounding skin. In well-marked cases the elevation may amount to tw or three millimeters, so that the patches stand out in bold relief. Th color varies through all the tints of inflammatory red; the patches ar usually pink; when, as occasionally happens, the process is more acut they may be a bright red; occasionally they show a violaceous tint. Th scales are fine and of grayish to grayish-yellow color. They are rathe adherent, but can be detached without bleeding. The scaling is rathe scant and is most pronounced usually over the border. In addition t the scales there are dirty grayish masses of sebaceous matter and epithelium which fill the dilated follicular openings. These, along with th scales, may be sufficient to give the entire patch a grayish tinge wit little evidence of the inflammatory base showing through except at th border, but ordinarily the appearance of the patches is that of a subacut sharply circumscribed, pink inflammatory infiltration whose color is ver slightly obscured by a scant covering of grayish scales.

The lesions of lupus erythematosus always maintain a uniform typ They are always dry, inflammatory patches with a tendency to scalines They never spontaneously become moist, and they never ulcerate. A the lesions spread peripherally the process may remain uniform through out the patch so that the infiltration and elevation, the redness, and th scaling are uniform over the surface; but usually there is some tendency to subsidence of the central area, so that the process is more active at the border and the central area is slightly depressed and shows the other features of a less active process. After reaching a certain size and showing this slight tendency to central subsidence, the lesions may remain indefinitely without change, or after a long or short time may undergo spontaneous involution. Upon disappearing they may leave no trace in the skin, but typically upon disappearance of the lesion there remains a thin, atrophic, slightly depressed, permanent scar, which from pitting at the follicular orifices has a sievelike appearance. These scars are soft and flexible with no tendency to contract, and produce no distortion. On the scalp they are apt to be somewhat thicker and firmer.

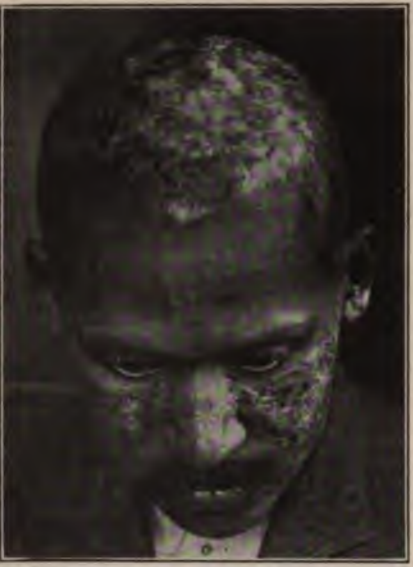

Fig. 94.—Lupus Erythematosus in Negro, showing characteristic distribution on face and scalp. (Pardee's case.)

The lesions are usually multiple upon the affected areas. In size they vary from a millimeter to several centimeters in diameter. They are usually the size of a shilling or less, but the coalescence of adjacent patches is common, and there are thus formed larger polycyclic patches. The lesions tend to occur in groups of symmetrical distribution, and have a very strong predilection for certain parts: the center of the face, the auricles, the scalp, and less frequently the backs of the hands. They may appear anywhere on the face, but the characteristic distribution is over the dorsum of the nose and the center of the cheeks, forming the so-called butterfly or saddle-shaped patch. The lesions here may coalesce until the saddle-shaped patch is one confluent lesion, but more frequently the eruption is made up of multiple lesions included in this distribution. Not infrequently the disease occurs on the face as a single patch involving the lower two thirds of the nose and spreading to some extent upon the adjacent parts of the cheeks. Along with these patches on the nose and cheeks it is usual to find the eruption also on the ears and on the scalp. On the scalp the lesions are apt to be without elevation and to occur as glistening, sharply circumscribed red patches which are permanently bald. Occasionally the disease occurs upon the scalp without appearing upon other parts. On the ears the lesions show the ordinary characteristics. Upon the helices they are apt to be less sharply defined and less infiltrated; in the concha they are more typical and show an abundance of plugged sebaceous follicles. Lupus erythematosus is not common on the hands.[1] When occurring there it is usually found on the dorsal surfaces of the hands and fingers, but it may spread to the palmar surfaces. The lesions are apt to be less well defined than upon the face, without scaling or slightly scaly, and of darker red to violaceous tint.

Occasionally lupus erythematosus appears upon mucous membranes[2] contiguous to involved skin, and lesions may be found on the vermilion border of the lips, in the mouth, in the nose, or on the palpebral conjunctiva. Thomas Smith,[3] from an examination of fifty-six consecutive cases, found mucous membranes involved in sixteen cases (twenty-eight per cent), an unexpectedly large proportion. On the mucous surfaces the lesions appear as bright-red or grayish-red macules or patches, sometimes definitely infiltrated, but usually flat and sometimes with a tendency to atrophy. It is doubtful if the lesions ever occur upon the mucous membranes without involvement of the skin, and under such conditions diagnosis would be impossible.

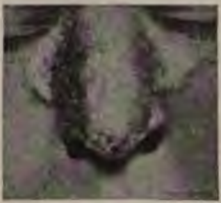

FIG. 95.—LUPUS ERYTHEMATOSUS. (Author's collection.)

The disease as a whole pursues a capricious and indefinitely prolonged course. While on the one hand lesions disappear spontaneously, on the other new lesions are apt to appear without apparent cause, and so the disease is prolonged for years. At times it may improve very greatly and apparently disappear, and then without discoverable cause flare up again.

The subjective symptoms of lupus erythematosus are usually so slight as to be of no consequence. There may be slight itching or feeling of tension about the lesions, and when the patches are temporarily more active the itching may be considerable. In the usual type of cases there are no constitutional symptoms.

Variations.[4]—In rare cases there appear in the disks of erythematous lupus round infiltrations which suggest tubercles of lupus vulgaris.

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[1] Hyde, *Jour. Cut. Dis.*, 1884, p. 321.—Klotz, *ibid.*, 1888, pp. 50 and 90.—Ohmann-Dumesnil, *Amer. Jour. Med. Sci.*, December, 1888.

[2] Fox, G. H., *Jour. Cut. Dis.*, 1890, p. 24.—Lustgarten, *ibid.*, 1897, p. 529.—Rille, *Wien. klin. Wochenschr.*, 1898.—Hassler, *Jour. des Mal. Cutan.*, January, 1900.—Bowen, *Twentieth Century Practice*, vol. v, p. 698.—Dubreuil, *Annales*, 1901.

[3] *Brit. Jour. Derm.*, vol. xviii, February, 1906, p. 59.

[4] Warde, *Brit. Jour. Derm.*, June, 1903.—Audry, *Jour. des Mal. Cutan. et Syph.*, 1904.

They are in most cases probably nothing more than points of denser infiltration of the process of erythematous lupus, but they at times show a picture very suggestive of a mixed type of lupus vulgaris and lupus erythematosus, and it is possible that the symbiosis may occur. These cases constitute the *lupus erythemato-tuberculeux* of Besnier and the *lupus érythématoide* of Leloir.

Nodular Erythematous Lupus.—Crocker describes a nodular form of erythematous lupus in which, perhaps in association with typical discoid patches, numerous round or oval elevated nodules from the size of a hemp seed to that of a bean appear scattered over the upper part of the face. These lesions are very like nodules of lupus vulgaris, but Crocker places them with lupus erythematosus on account of their general behavior and their distribution.

Telangiectatic Erythematous Lupus.—In this rare form of the affection the peculiar lesions are usually limited to the face, and occur as persistent circumscribed patches of erythema on the flush areas of the cheeks, the erythema being due chiefly, as can be seen upon close inspection with a hand lens, to numerous dilated vessels. There is an absence of scaling and usually of comedones, and the patches are flat, but they are infiltrated and may be elevated and œdematous. They may persist for years with little alteration, but when involution occurs it is followed by distinct atrophic scarring. These vascular patches may be associated with discoid lesions on the scalp.

Lupus Erythematosus Disseminatus.[1]—In rare cases lupus erythematosus becomes generalized. The lesions develop widely and rapidly over the various parts of the body, and in extreme cases become almost universal. In such cases the lesions are of more acute inflammatory type and there is more crusting and scaling, so that the disease may resemble a dry, scaly, or a crusted eczema, but the borders are well defined and more or less infiltrated, and upon removing the crusts the plugs extending into the sebaceous follicles and the gaping follicles are exposed. These cases usually pursue an acute course and are accompanied by fever, joint and bone pains, and gastrointestinal symptoms—constitutional symptoms such as are seen in erythema multiforme and suggesting a similarity in the etiology of the two affections. Such attacks may be the primary manifestations of the disease or they may supervene upon the typical form of it. Repeated acute attacks of this sort may occur. The cases are, however, excessively rare.

Cases have been recorded by Besnier, Hallopeau, Kaposi, and others, which appear as polymorphous erythemas, sometimes with vesicles and bullæ, and which subsequently assume the clinical appearances of disseminate erythematous lupus. These are probably borderline cases between lupus erythematosus and erythema multiforme. Short [1] has recorded a case of this sort in a woman of twenty-eight which was fatal after six months' illness.

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[1] Kaposi, *Archiv*, 1872, p. 36.—Hardaway, *Jour. Cut. Dis.*, 1889, p. 448, and 1892, p. 268.—Koch, *Archiv*, vol. xxxvii, 1896, p. 39.—Cavafy, *Brit. Jour. Derm.*, 1897, p. 328.—Bulkley, *Jour. Cut. Dis.*, 1897, p. 178.—Brooke, *Brit. Jour. Derm.*, 1895, p. 73.—Jamieson, *ibid.*, 1893, p. 115.

Erysipelas Perstans Faciei.—Under this name Kaposi has described a condition which occurs as persistent erysipelas-like swellings of the face, accompanied by temperature reaching 104° F. and typhoid symptoms, followed by coma and, in fifty per cent of the cases, death. Its nosological position is very uncertain.

Lupus Pernio.—Occasionally lupus erythematosus upon the ears and hands follows chilblains. The lesions around the borders of the ears may be indistinguishable from the ill-defined purplish inflammatory patches of chilblains. Later they may assume, especially in the concha, the typical appearance of discoid patches of lupus erythematosus. This form of lupus erythematosus on the ears and also frequently on the hands, usually without involvement of the typical areas of the face or of the scalp, is known as lupus pernio or chilblain lupus.

Lupus Livido.—This term is applied to lesions occurring on the extremities of patients suffering from peripheral asphyxia. They occur as persistent purplish patches which eventually may ulcerate. They are phenomena of local asphyxia, the extreme type of which are seen in Raynaud's disease, and are not a form of lupus erythematosus.

Complications.[2]—Hutchinson has described an acneiform eruption over the shoulders associated with long-standing lupus erythematosus. Crocker refers to a case of Galloway's with lupus erythematosus of the face in which, during a period of six weeks, the scalp lesions developed bullæ. Crocker, Hallopeau, and others have seen follicles of the hands in association with lupus erythematosus.

The development of epithelioma in patches of lupus erythematosus has been recorded by Pringle,[3] Dyer,[4] and others.

Etiology.—The cause of lupus erythematosus is unknown. It usually develops between eighteen and forty years of age, with extreme limits of three years (Kaposi) and seventy-one years (Crocker.) It is three to five times as common in women as in men. Unlike lupus vulgaris it is in the majority of cases seen in women of the well-to-do class. It is said to be commoner in persons of light hair and fair skin, but I have seen a severe discoid case in a full-blooded negro. Sequeira and Rona have both reported cases in sisters.

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[1] *Brit. Jour. Derm.*, August, 1907.

[2] Bornemann, *Dermatolog. Zeitschrift*, May, 1905, p. 349.

[3] *Brit. Jour. Derm.*, 1900, p. 1.

[4] *Daniel's Texas Med. Jour.*, 1892–93, vol. viii, November, 1892, No. 5, p. 178.

There is impressive evidence in favor of its association with tuberculosis. Boeck [1] in 42 cases found evidence of past or present tuberculosis in 28; Roth [2] in 250 found similar evidence in 185. The disseminate form particularly is associated with tuberculosis. On the other hand, lupus erythematosus is frequently seen in individuals giving no history and showing no evidence of tuberculosis, so that tuberculosis can be regarded as only one of its causes. When due to tuberculosis it may be a manifestation of tuberculin toxæmia, as it probably is in many other cases a manifestation of other forms of toxæmia. Sequeira and Balean [3] have found albuminuria in half the cases and Crocker believes that the association with copious albuminuria is too frequent to be a coincidence. Occasionally it is associated with uterine conditions. Fordyce has reported a disseminate case and refers to a discoid case which disappeared during pregnancy. I have had a typical discoid case of long standing which disappeared completely within two weeks after an operation for uterine myoma. Various depressing conditions, such as chlorosis and anæmia, digestive and nervous disturbances, are apparently at times factors in its etiology, and are often of definite influence in exaggerating the disease. It occasionally follows smallpox, scarlet fever, erysipelas, and superficial inflammatory diseases of the skin. A feeble peripheral circulation is apparently at times the cause of its development. The influence of cold in its production has been referred to under lupus pernio, and the disease also at times follows exposure to extreme heat. Anything, in fact, which tends to impress deeply the peripheral vascular tone may be a factor in its excitation.

The mere recital of its possible causes indicates how complex the etiology of the affection is. It is probably an angioneurotic process which may result from various causes: from toxæmia, from reflex nervous disturbances, perhaps from nervous disturbances of central origin, or from disturbances of the local vasomotor mechanism, as, for example, when this has been permanently impressed by great exposure to cold or heat.

Pathology.[4]—There has been much disagreement among the various students of the pathology of lupus erythematosus in regard to both the histological facts and their interpretation. The consensus of opinion can be summarized in the statement that the process is one of localized chronic inflammatory reaction of peculiar character and associated with a vasomotor disturbance. No affirmative pathological evidence of a tuberculous nature of the disease has been found; tubercle bacilli have not been discovered, either in the lesions or by inoculations, and the histology bears no similarity to that of a tuberculous process.

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[1] *Archiv*, 1898, vol. xlii, p. 71.

[2] *Archiv*, 1900, vol. li, p. 3.

[3] *Brit. Jour. Derm.*, 1902, vol. xiv, p. 367 (Bibliography).

[4] Robinson, *Trans. Amer. Derm. Assn.* for 1898.—Fordyce and Holder, *Med. Record*, New York, July 14, 1900.—Schoonheid, *Archiv*, 1900, vol. liv, p. 163.—Audry, *Jour. des Mal. Cutan.*, 1901.

The principal anatomical changes occur in the upper half of the corium. There is great distention of the capillaries and small vessels and of the lymphatics, the blood vessels being filled with red and white blood cells, while around them and the lymphatics and within the latter is an infiltration of plasma cells, which are usually tightly packed and of uniform size; multinucleated and giant cells are absent. The infiltration may be slight and confined to the vicinity of the vessels, or may invade the deeper corium and subcutaneous tissue, or involve the whole depth of the corium, penetrating the sebaceous glands and the ducts of the sweat glands, and thus causing their subsequent atrophy. The cells of the infiltration undergo no degeneration *en masse*, but individually gradually undergo atrophy and absorption. Fordyce and Holde have shown that in recent lesions the capillaries are blocked, and the consider that a localized capillary stasis with increased pressure is th primary lesion of the disease. The collagenous bundles in the vicinit of the infiltration early show a peculiar degeneration, having a cloud appearance and staining dark brown with acid orcein; later they also t a large extent undergo absorption. The elastic tissue for the most par is unaffected.

The changes in the epidermis consist in the early stages of œdem and hyperkeratosis, and later of atrophy of the rete mucosum and of th glandular structures. These are by most observers considered purel secondary, though Unna and Schoonheid think them primary.

Diagnosis.—The characteristic features of lupus erythematosus ar the discoid inflammatory patches with sharply defined borders; the sligh scaliness associated with sebaceous plugs dipping into the gaping folli cles; the symmetry and distribution on the cheeks, nose, ears, an scalp; the absence of ulceration, papules, and nodules, and the presenc of slight atrophic scarring; the age at which it appears, and its indolen persistent course. The picture which it produces is usually so characte istic as to make mistake in diagnosis inexcusable. On the other hand, i the variant cases and in the acute disseminate cases diagnosis may b very difficult.

Lupus vulgaris is distinguished from it by the presence of the cha acteristic apple-jelly tubercles, by ulceration and serious destruction, b the formation of fibrous scars, and by its appearance in childhood.

A diffuse patch of nonulcerating serpiginous syphilide upon the nos may superficially resemble erythematous lupus. This, however, is com posed of aggregated tubercles which give the surface a nodular contour. As the result of their involution small pea-sized, pitted, depressed scars are formed. The nodular character of the patch is well made out at the border, which is also less sharply defined than that of lupus erythematosus; the course is more rapid, and there is entire absence of the gaping plugged follicles.

The disease may also be confused with acne rosacea, seborrhœic dermatitis, chronic scaling eczema, and tinea circinata, but the resemblances are so superficial that a consideration of the characteristic features of these different affections makes the diagnosis easy.

Prognosis.—Erythematous lupus is extremely capricious in its course and very rebellious to treatment, and definite assurance of permanent relief cannot be given. Aside from the superficial atrophic scarring it causes no destruction of tissue, and, except for the disfigurement from the presence of the patches, no serious inconvenience. The rare acute disseminate forms and the erysipelas-like form of Kaposi may endanger life.

Treatment.[1]—The internal treatment of lupus erythematosus is symptomatic, and usually altogether without influence upon its course. Various remedies have been suggested upon empirical grounds: potassium iodide, iodoform, iodide of starch, salicin, and quinine in large doses, ichthyol, phosphorus, arsenic, and the various remedies which reduce cutaneous hyperæmia, among which ichthyol is supposed to hold first place. No reliance can be placed upon any of these. Salicin and quinine are sometimes definitely beneficial.

In the local treatment the greatest benefit is to be gotten from the use of soothing and astringent or slightly irritating applications. Only after these have been persistently used and after the cases have been carefully watched should recourse be had to vigorous applications, and radically destructive applications are never to be relied upon. When areas are acutely inflamed they should first be treated with soothing applications, like calamine lotion, or bland salves until they become less irritable. In the usual type of subacute cases it is a good course first to wash the areas with green soap and water once daily, producing slight but not pronounced irritation. Along with this measure the frequent application daily of calamine lotion is often very beneficial. In place of this or as a substitute for it when the lesions become less active, an excellent application is lotio alba, from 5 to 15 grains potassium sulphide and zinc sulphate to 4 ounces of water. Other preparations of sulphur are also frequently useful—for example, ½ to 1 dram precipitated sulphur to the ounce of ointment. In place of sulphur, ichthyol under the advocacy of Unna is recommended; in my experience it has no advantages, and is unsightly and disagreeable.

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[1] White, J. C., *Jour. Cut. Dis.*, 1898, p. 457.—Unna, *ibid.*, 1898, p. 465.—Brocq, "Traitment des maladies de la Peau."—Crocker, *Brit. Jour. Derm.*, 1898, p. 8.—Whitehouse, *N. Y. Med. Jour.*, vol. lxix, 1899, p. 159.—Hebra, Hans, *Wien. med. Wochenschr.*, 1899, p. 14.—Bukovsky, *ibid.*, 1899, pp. 1450 and 1500 (review of treatment).

When patches are much infiltrated or of very indolent character, they may be treated more vigorously by various stimulating methods. Among the best of these is vigorous scrubbing with green soap to the point of producing redness and a little bleeding of the surface. Another plan is to dissolve off the scales and some of the horny epidermis by applications of liquor potassæ. Or the same result may be obtained by the use of salicylic acid, 10 to 20 grains or more in ointment, or by its application in the strength of two to six per cent in collodion, or by covering the patches with mercurial plaster until they become irritated. Under any of these various methods the treatment is kept up until the lesions become irritated, and then soothing applications like calamine lotion ar applied until the irritation entirely subsides, when the vigorous metho of treatment may be tried again. At times plain collodion may be ap plied to the lesions with advantage, purely for its mechanical effect i compressing the capillaries. Hans Hebra highly recommended the use o absolute alcohol or alcohol and ether for the purpose of removing th fat and moisture from the surface of the lesions. The method has prove disappointing.

Of the more vigorous methods of treatment which are occasionall to be tried, one which offers good prospect of benefit is the paintin as suggested by G. H. Fox, of a few patches at a time with pure carbol acid. Other vigorous applications which are used are resorcin (thr to ten per cent) in collodion, sodium ethylate or chloracetic acid car fully painted on, and ten per cent pyrogallic-acid ointment. All of the are used to the point of producing dermatitis or a slight eschar, wh they are followed by bland ointments like boric-acid vaseline. All these methods of treatment which produce violent reaction or whi cause destruction of tissue should be used with great caution and ov small areas, for the reason that they are apt to cause considerable sca ring and at the same time may not cause the disappearance of t patches.

In the absence of satisfactory results from the older methods of trea ment, there has been a general attempt recently at the treatment of the cases with x-rays, ultraviolet light, and high-frequency currents. The methods are all occasionally successful, but none of them is entire reliable. Schiff, Stelwagon, Hyde and Montgomery, and various othe have had favorable results from Roentgentherapy and phototherap Frequently cases can be quite cleaned up under careful exposures x-rays, but the results are by no means always certain, and in many cases there is practically no benefit. The same is true of the use of the Finsen light and of high-frequency currents. The high-frequency current applied daily or every other day to the point of producing temporary erythema has succeeded in some cases.

Many dermatologists are impressed with the results obtained by freezing the patches with solidified CO_2 or liquid air (in principle the same thing). My results with this method, while not uniformly successful, have been so favorable as to lead me to believe it is by far the most effective treatment we have in the ordinary chronic forms of lupus erythematosus. My method is to freeze the patches for ten to twenty seconds at intervals of at least three weeks; others have reported to me good results from freezing up to sixty seconds. As a rule, one or two freezings leave smooth white scars, and I have seen not the slightest ill effect. I have not used it in pernio or acute cases.

II.—DERMATITIS SEBORRHOICA[1]

SYNONYMS: *Eczema seborrhœicum, Seborrhœa corporis, Pityriasis capitis, Seborrhœa sicca.*

Seborrhœic dermatitis is a disease of the skin, probably due to a specific organism, characterized by the occurrence of patches of subacute dermatitis which are associated with scaling and hypersecretion of fat.

The condition which we now recognize as seborrhœic dermatitis was formerly known as several distinct affections: pityriasis capitis, seborrhœa sicca, seborrhœa corporis (Duhring), lichen circumscriptus (Willan and Bateman), lichen annulatus (Wilson), lichen gyratus (Biett and Cazenave). These are all characterized by the presence of more or less greasy scaliness, usually upon an inflammatory base. Van Harlingen first showed that pityriasis capitis was not a true seborrhœa; and, before Unna, Duhring described seborrhœa corporis, and thus indicated the identity between the affection as it occurs upon nonhairy parts of the body and upon the scalp and face. But to Unna belongs the credit of having made the generalization which includes all of these affections under one conception, and of having established their essentially identical character. Unna's original conception of seborrhœic dermatitis, announced in 1887, like most other generalizations, included t but, subject to certain eliminations, it has borne the test of ti there are now few authorities who doubt either that the various which Unna brought together are forms of one disease or that se dermatitis is a distinct disease.

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[1] Unna, "Histopathology," *Monatshefte*, vol. vii, 1887; *Jour. Cut. and Gen.-Urin. Dis.*, 1887; *Brit. Jour. Derm.*, 1894.—Elliot, *N. Y. Med. Jour.*, vol. liii, 1891; Morrow's "System," vol. iii; *Jour. Cut. and Gen.-Urin. Dis.*, 1893.—Van Harlingen, "A Contribution to the Pathology of Epithelium," *Amer. Jour. Med. Sci.*, 1876; "Pathology of Seborrhœa," *Arch. Derm.*, 1878.—Duhring, Part II, p. 323.—Sabouraud, "Bacteriology of Seborrhœic Dermatitis," *Bull. Pasteur Institute*, No. 2, 1897.—Merrill, *N. Y. Med. Jour.*, vol. lxii, 1895, and vol. lxv, 1897.—Whitfield, *Brit. Jour. Derm.*, 1900.

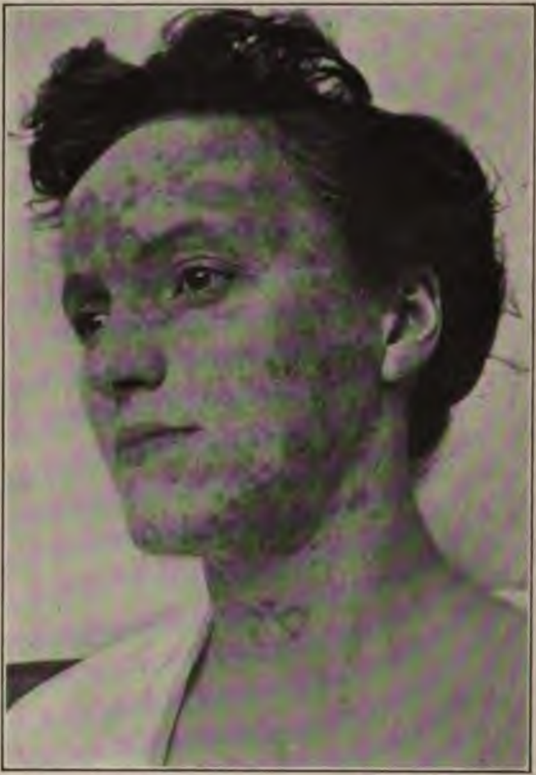

FIG. 96.—ECZEMA SEBORRHOICUM IN CIRCINATE PATCHES. (Schamberg's collection.)

Seborrhœic dermatitis is not a form of ordinary eczema, and that it may not be confused with ordinary eczema it seems bett the term derm orrhœica, as by Elliot, rat Unna's origin eczema seborrh

Symptom —Seborrhœic tis in all of i consists of a s inflammatory usually subacu is accompanie ing and an accumulation upon the surf its slightest m tions it may sh area more or ered by grea without redd the base or p evidences of i tion. The pat resenting the gree of the di show the greas scaliness, but neath this there is a reddened, perceptibly inflamed base. In it development there is a well-defined, subacute inflammatory pro the production of light to dark red, slightly infiltrated patche result of secondary factors, as, for example, the maceration of th in the axillæ, the process may become acute with the formation o but seborrhœic dermatitis of itself does not become of sufficie sity to produce a weeping dermatitis.

In the slighter degrees of seborrhœic dermatitis where th scaliness is the chief manifestation and the evidences of infla are very slight or absent, the borders of the patches may be ill defined. Except in these cases the patches are sharply circumscribed and oval or round. They vary from scaling maculo-papules to lesions the size of a finger nail or a coin or larger. The patch usually develops as a whole and not by the enlargement of a small lesion by peripheral extension. Frequently the process is active over the entire area of the lesion, so that there is a diffuse subacute dermatitis over the whole of the surface. With equal frequency, however, the lesions show a tendency to central involution, and then there are formed circinate lesions, the centers of which may be normal but usually present only a less active manifestation of the process.

The greasy scaliness of the patches is the most characteristic feature of the lesions. The scales are whitish, or more frequently yellowish, and from the admixture of fat are soft and greasy. They may be thin and scanty, or they may be abundant and cover the surface in a soft, rather thick layer. They are not closely adherent, like the scales of psoriasis. They are particularly abundant upon the scalp and other hairy surfaces. Upon nonhairy surfaces upon which there is free perspiration, or which are exposed to the friction of clothing, and in persons who bathe a great deal, the amount of scaliness may be very slight. Under such circumstances the patches show as rather glistening, unctuous, reddish areas of subacute dermatitis with slight exfoliation of thin whitish scales. When the scales are abundant, upon their removal the epidermis is thin and glistening, perhaps showing weeping points, and occasionally the entire surface may be moist. Weeping lesions, however, are quite exceptional.

The extent of seborrhœic dermatitis varies greatly. It begins always upon the scalp, and in most cases is confined to the scalp. It also appears upon the face and neck, the chest, especially over the sternum, the back, especially in the interscapular space, and less frequently it involves the umbilicus, the axillæ, the inguinal region, or becomes generalized over the trunk and extremities.

On the scalp the most common manifestation of seborrhœic dermatitis is the condition which we know as dandruff. There may be little oily secretion, and then the process manifests itself as diffuse patches more or less completely covered with fine branny scales, some adherent to the scalp, others loose in the hair; this is the condition known as pityriasis capitis. Or the scales may be more abundant and accumulate in thick, adherent masses of whitish greasy scales, the so-called seborrhœa sicca. In such cases the hair is dry and lusterless. More frequently there is an excess of oily secretion, the hair is smooth and oily, and the scales are soft and greasy and yellowish. When the scaliness is very slight, as in pityriasis capitis, there may be no macroscopic evidences of

inflammation, although microscopically there are seen in the corium
changes of a superficial inflammatory process of slight intensity. W
the scaliness is abundant and where there is much crusting, the pat
are usually of pinkish or red color, slightly infiltrated, and dry, but o
sionally show points of excoriation. These more pronounced pat
on the scalp usually show the sharply defined border of the patches
nonhairy parts, and frequently the central involution and the tend
to the formation of gyrate and circinate lesions which is seen elsew

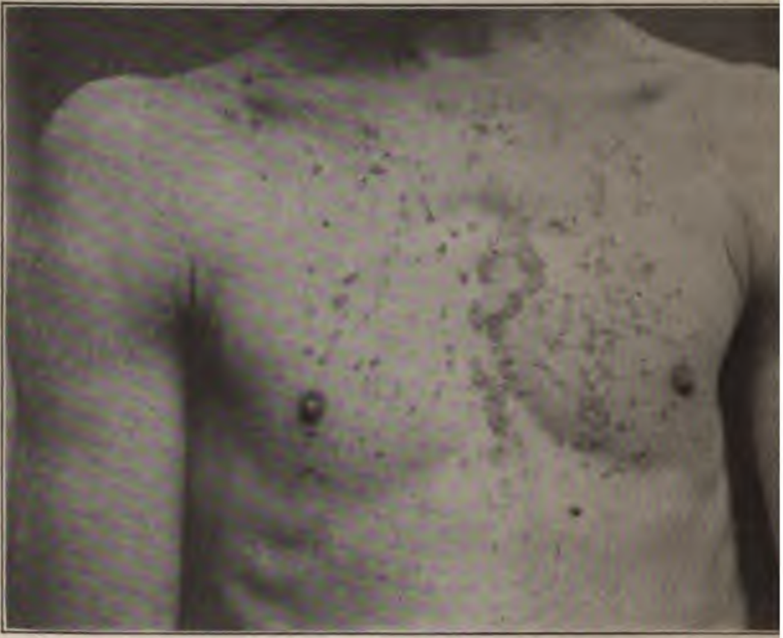

Fig. 97.—Eczema Seborrhoicum. (Schamberg's collection.)

The disease not infrequently spreads beyond the border of the
scalp. It appears upon the forehead as areas of subacute dry derm
of pinkish to reddish color, usually slightly scaly, occasionally co
with scales. Whether the scaling is abundant or slight, these pa
on the forehead, as elsewhere upon nonhairy parts, have a soft, unc
feel. The borders of the patches on the forehead are usually shar
of circinate outline, so that the confluence of adjacent patches give
entire eruption a polycyclic border. In the same form the eruptio
quently spreads on the nonhairy skin behind the ears and down o
neck.

Next to the scalp the process most frequently shows itself o
face—around the nose, on the ears, in the eyebrows, and on the

lip, and less frequently in the beard. In the eyebrows, on the upper lip, and in the beard it appears usually with the formation of an abundance of greasy yellowish scales under which there are diffuse areas of dry dermatitis. From the eyebrows it may spread to the eyelids. The ears are frequently involved. As it involves the ears, it does not present any essential differences from its appearance upon the forehead. It very frequently appears in the concha and at the auditory meatus without involving the rest of the auricle. It may show here simply as a greasy scaliness with diffuse redness, or it may occur in sharply circumscribed inflammatory patches covered by greasy yellowish scales. Either with or without involvement of other parts of the face a form of seborrhœic dermatitis of slight intensity frequently involves the nose, especially the alæ, the naso-labial folds, and the adjacent parts of the cheeks. In this form of the disease the involved skin is greasy, more or less scaly, of a muddy yellowish tinge, or pinkish or reddish, but without definite infiltration, and the sebaceous follicles are unduly prominent. This form of seborrhœic dermatitis is frequently responsible for moderate types of red nose, those types which are reddest in the naso-labial fold with diffuse oiliness of the surface. Occasionally in this distribution sharply circumscribed infiltrated areas of seborrhœic dermatitis occur, but they are not nearly so common as the ill-defined diffuse erythematous areas just considered.

In rare cases the vermilion borders of the lips are affected, producing the condition known as *cheilitis exfoliativa.*[1] The lips are inflamed, somewhat inelastic with more or less fissuring, and covered with adherent scales or crusts. The process may be sharply limited to the vermilion border, but it is usually associated with greasy scaliness of the nose and with seborrhœic dermatitis of the scalp. It is the most intractable form of seborrhœic dermatitis.

As seborrhœic dermatitis occurs upon the trunk it shows a distinct predilection for the median lines of the chest and back. Its most frequent sites of development on the trunk are the chest over the sternum and the interscapular space; thence it spreads laterally. This predilection for development in the median line and for lateral spread is a characteristic feature of importance in the diagnosis of seborrhœic dermatitis from psoriasis, whose predilection is for bilateral distribution with spread *toward* the median line. Over the sternum seborrhœic dermatitis usually occurs in sharply defined round or circinate or gyrate patches. The patches may show a uniform inflammatory infiltration, or there may be central involution with an active border. Over these patches there is the usual yellowish greasy scaliness. On the back there is more frequently the occurrence of the diffuse pinkish patches with greasy scaliness. In and around the umbilicus the disease frequently occurs, showing the same characteristics as upon the sternum. Less frequently the axillæ and the inguinal folds are involved. In these locations the disease may occur as diffuse inflammatory patches with sharply circumscribed borders, or in lesions of gyrate or circinate outline with more or less involution of the centers. If the most active areas of the disease are not acutely inflamed they show the ordinary characteristics of the patches as they occur elsewhere, but in these locations from warmth and maceration the inflammatory process is apt to be more acute and the active areas are likely to be moist or weeping.

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[1] Stelwagon, "A Report of Two Cases of Persistent Exfoliation of the Lips," *Jour. Cut. and Gen.-Urin. Dis.*, 1900.—Kaposi, Besnier-Doyon's Edition, vol. i, 1891. —Unna, *Monatshefte*, vol. xi.—Galloway, *Brit. Jour. Derm.*, vol. vii, 1895.—Jamieson, *Brit. Med. Jour.*, December 7, 1895.

Instead of confining itself rather sharply to such locations as have been described, seborrhœic dermatitis occasionally becomes widely generalized over the trunk and extremities. When thus generalized it may be associated with large circumscribed lesions of the usual type in the areas of predilection, and with occasional large lesions upon other surfaces, but for the most part the generalized eruption consists of lesions from the size of a tackhead to a finger nail, pinkish or reddish in color, and more or less covered by whitish scales which upon surfaces without coarse hairs may lack the characteristic greasy feel. These patches may be rather ill defined in outline or they may be sharply defined, and in shape may be round or show some tendency to ring formation. In the well-marked generalized forms of seborrhœic dermatitis the process may rather closely simulate psoriasis, except in distribution.

Seborrhœic dermatitis is described as occurring on the palms and soles, either as diffuse scaling patches or as sharply circumscribed scaling patches of chronic eczema. These patches cannot be differentiated from ordinary eczema of these parts; their occurrence in association with seborrhœic dermatitis upon other nonhairy parts is very uncommon; and in my opinion it is extremely doubtful if these patches on the palms are a form of seborrhœic dermatitis.

Seborrhœic dermatitis—after heredity—is the most frequent cause of baldness. In pityriasis capitis the hair may be dry and lusterless; in the more pronounced degrees of seborrhœic dermatitis of the scalp the hair is usually oily and glistening. As the disease continues year after year there is gradual thinning of the hair, usually beginning over the vertex where the process is most intense. There is perhaps no diminution in the number of hairs, but as the hairs regrow they become shorter and finer, and ultimately from destruction of the follicles by the chronic inflammatory process baldness results. This, of course, is a matter of years.

The course of seborrhœic dermatitis is persistent. Lesions upon non-hairy parts disappear spontaneously or readily yield to treatment. On the scalp the disease persists indefinitely, and after apparent cure is likely to recur.

Seborrhœic dermatitis is essentially indolent, and is accompanied by little irritation. There is slight itching and burning, but this is never enough to be more than an annoyance. When from any cause an acute dermatitis supervenes upon the condition there is, of course, the itching of ordinary dermatitis.

Complications.—So persistent a condition as seborrhœic dermatitis is apt to be a frequent complication of transient dermatoses, but the complication is not likely to mask either condition. Seborrhœic dermatitis and acne upon the face frequently occur at the same time, but the appearance of neither condition is altered to any considerable extent by the combination. The association with papular syphilides of the face is referred to under syphilis, and is a common symbiosis. It gives to the ordinary scaling papular syphilide of the face a greasy yellowish appearance.

Etiology.—Seborrhœic dermatitis of the scalp is one of the commonest of diseases. Indeed, if we include in it, as I think should be done, the ordinary cases of greasy dandruff, a large majority, at least of the better class of urban population, has the disease. As it occurs on the face and over the chest it is a very common affection. The other forms of seborrhœic dermatitis are not nearly so frequent.

It occurs at all ages, perhaps most frequently in early adult life; in both sexes indifferently; and, unlike many dermatoses, is no respecter of the better classes. Impairment of health and disturbance of functions, such as constipation and other gastrointestinal disturbances, menstrual disorders, anæmia, tuberculosis, and other cachectic conditions are said to predispose to it. This may be true, but certainly the disease is seen constantly in individuals of normal health; and treatment directed to the improvement of the bodily vigor exerts practically no influence upon it.

Local influences are important in its production. Lack of attention to cleanliness of the scalp is a predisposing factor in its etiology, as is carelessness about bathing and keeping the body free from the accumulation of sebum, exfoliated epidermis, and perspiration. The greasy, oily, freely perspiring skin seems to be predisposed to the condition. A sedentary mode of life and life indoors predispose to it, and it is more frequent in winter than in summer. The disease is very probably spread by infection, and the common use of combs and brushes, especially in barber shops and other public places, is probably the usual means of its diffusion.

Pathology.—The characteristic histological features of seborrhœic dermatitis are pronounced vascular dilatation and œdema of the corium, and intra- and intercellular œdema of the epidermis, with hyperplasia of the rete mucosum. They are very similar to those of psoriasis. There is dilatation of the vessels of the corium, especially of the papillary layer, with œdema and leucocytic infiltration and occasionally round-cell proliferation around the vessels and glandular structures. The papillæ are lengthened and often appear smaller in diameter, an appearance due to the hyperplasia of the prickle-cell layer. The basal layer of the epidermis remains intact, but the interpapillary processes are increased in both depth and thickness. There is marked œdema of the epidermis; the cells are swollen and perinuclear spaces ar visible. The intercellular space are greatly dilated, the prickle stretched to the full extent, an between the cells occasional leuc cytes may be seen.

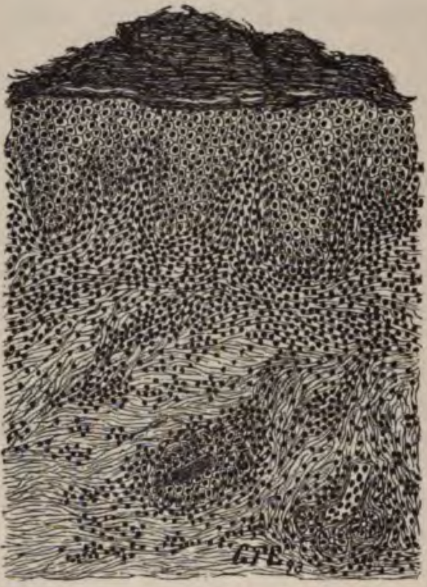

FIG. 98.—A SMALL PAPULE OF SEBORRHŒA CORPORIS. In the cutis dense inflammatory infiltration. (G. T. Elliot, Morrow's "System.")

Unna attributes the vascul dilatation and œdema to the irr tation of a microörganism, h morococcus, and Merrill claims have reproduced the disease by i oculation of pure cultures of th organism. The morococcus is a mulberry-shaped staphylococcus foun often in pure cultures in the tissues, and producing erythema, vesicle and subsequent alopecia when inoculated in rabbits. Unna's views ha not found general acceptance, and the bacteriology of the disease is yet uncertain. It is, indeed, probable that Unna's morococcus is t common staphylococcus epidermidis albus of Welch.

Diagnosis.—The characteristic features of seborrhœic dermatit are the greasy, unctuous character of its lesions, its occurrence up the scalp and its characteristic distribution upon other parts, the usu sharply circumscribed, rounded borders of the patches, and the slig degree of itching. These features all sharply differentiate it from ord nary eczema.

A few circinate patches may be confused with ringworm, but rin worm has no such typical locations, there is no greasy character to th scales, the inflammatory process is apt to be more acute, and the fungus can be found in the lesions. On the scalp there is entire absence of the bald or partially bald patches, and of the twisted and broken and friable hair stumps which characterize ringworm. Ringworm of the genitocrural region and seborrhœic dermatitis of the same location may closely simulate each other, and a differential diagnosis may depend upon the determination of the presence or absence of the ringworm fungus. Genitocrural ringworm, however, is usually a much more acute inflammatory process than seborrhœic dermatitis.

Extensive cases of seborrhœic dermatitis occurring in the form of coin-sized, sharply circumscribed patches may resemble psoriasis very closely. The most marked difference between the individual lesions of psoriasis and seborrhœic dermatitis is in the character of the scales. The scales of psoriasis are dry, harsh, whitish, and closely adherent; the scales of seborrhœic dermatitis are greasy, soft, yellowish, and easily removed. Seborrhœic dermatitis begins on the scalp and hairy parts, shows a predilection for hairy surfaces, and is common on the face; it is usually most marked in the median line and diminishes laterally, while psoriasis begins on the elbows and knees as well as the scalp, very rarely involves the face, and is least abundant as a rule upon those parts of the trunk where seborrhœic dermatitis is most abundant. On the scalp seborrhœic dermatitis is usually diffuse, without sharply circumscribed patches, and the scales are greasy and soft, while the patches of psoriasis are circumscribed and thickly covered with scales, which retain their dry, harsh, whitish character. The body cases may resemble pityriasis rosea.

Prognosis.—Seborrhœic dermatitis, except on the scalp, yields readily to treatment. There is some tendency to recurrence upon the non-hairy parts, but this is not sufficiently pronounced to render the affection difficult to handle. On the scalp it is not difficult to get rid of the well-marked inflammatory lesions, but in the form of abundant greasy scaliness with or without slight inflammatory reaction the disease is extremely rebellious. It can easily be improved, but its eradication is difficult and it is almost impossible to avoid recurrence. This is explained frequently on the ground of reinfection, but, be that as it may, after the disease has once been thoroughly established it is very apt to persist indefinitely or recur repeatedly.

Treatment.—The treatment of seborrhœic dermatitis is for all practical purposes entirely local, and consists in the use of antiseptics. Of these the most useful is sulphur. Resorcin, suggested for this condition by Unna and highly recommended by Elliot, is largely used, but in my experience is very unreliable. It gives the hair a peculiarly harsh, disagreeable feel, and occasionally, unless chemically pure, produces a slight greenish-yellow discoloration of the hair, so that I have practically discontinued its use, although some of the formulæ containing it which are recommended by others are given.

Upon nonhairy parts ointments furnish the best form of application in seborrhœic dermatitis. Applications for the nonhairy parts must be more dilute than those which are used upon the scalp.

For the diffuse erythematous greasy areas of seborrhœic dermatitis about the nose the most useful application is, in my experience, lotio alba:

℞ Zinc sulphate, Potassium sulphide,	āā	gr. v–xv;
Water		℥i.

To this may be added 5 to 15 grains of sulphur.

This application should be dabbed upon the parts for two or three minutes several times daily, its action being kept just short of producing irritation. For the dryness of the skin which it causes after a few days, it is well to use an ointment of cold cream containing 5 grains salicylic acid and 10 grains sulphur to the ounce. Occasionally such ointments as are suggested for other nonhairy parts may be used to good advantage upon the face. Vleminckx's solution may be used in the same way as lotio alba. In connection with whatever antiseptic application is used the face should be washed at least once a day with an alkaline soap, such as tincture of green soap, to get rid of the grease and the loosened horny epidermis. This is best done at night just before the application of the medicament.

Upon acutely inflamed areas of seborrhœic dermatitis, as in the inguinal fold and axillæ, it is desirable to use first soothing lotions or ointments, such as are used in eczema.

In all the other forms of seborrhœic dermatitis upon nonhairy parts the ointments of sulphur combined, if you please, with the other commonly used remedies are satisfactory. The following are good formulæ:

℞ Acidi salicylici		gr. v–x;
Sulphur præcip.		gr. xx–lx;
Ung. zinci oxidi		℥i.

℞ Sulphur. præcip.		gr. xv–xl;
Ac. salicylici		gr. v–xv;
Pulv. amyli, Pulv. zinci oxidi,	āā	ʒiss;
Vaselini	ad	℥i.

Either of these should be applied freely twice daily and allowed to grease the clothing so that their slight application is constant.

On the scalp it is usual to recommend for daily application solutions of resorcin in water (one to six per cent), of salicylic acid (two to ten per cent) or of salicylic acid and resorcin (two to six per cent) in alcohol or equal parts alcohol and water; but these leave the hair with a disagreeable feel, and in my experience they are never curative. Their use must be combined with the more or less frequent use of ointments.

As an ointment for seborrhœic dermatitis of the scalp I have found nothing better than

℞ Salicylic acid	gr. x–xxx;
Sulphur	ʒss–ij;
Rose ointment or vaseline	℥i.

Special bases, such as Jackson's cream, are recommended, but they are difficult of preparation, and in my experience are not better than rose ointment. A pomade should be applied daily to the scalp; by parting the hair at successive points over the patches the ointment can with care be applied with little greasing of the hair. When after several days the hair has become uncomfortably greasy the scalp should have a shampoo. For this purpose tar soap is most satisfactory and most agreeable; next to this is tincture of green soap. A soap of resorcin and salicylic acid (each three per cent) and sulphur (ten per cent) which is obtainable in commerce is sometimes recommended, but has no advantages. After the markedly inflammatory lesions have entirely or in great part disappeared, the ointment may be applied to the scalp once or twice weekly, and in the intervals the scalp thoroughly wet once daily with an antiseptic solution. Resorcin and salicylic-acid solutions, as indicated above, are usually recommended. Instead of these I have found much more agreeable, and I believe equally or more effective, solutions of corrosive sublimate or of formalin (1:1,000 to 1:250) in alcohol or in equal parts alcohol and water. Indeed, I frequently recommend that the patient wet the scalp thoroughly once a day with alcohol alone, and have found it quite as satisfactory in keeping the condition cleaned up. Alcohol as a menstruum is particularly desirable in these cases, because it evaporates quickly and does not leave the scalp moist.

By the persistent use of such lotions combined with the occasional use of the ointment the cases can be carried along to apparent cure. After this the patient should be instructed to apply to the scalp several times a week either alcohol or one of the antiseptics in alcohol. Such a habit in my experience is the best assurance against recurrence of the disease.

In the treatment of seborrhœic dermatitis of the vermilion border of the lips I have found the ointments of salicylic acid and sulphur, such as are used on the body, the most satisfactory preparations. Under their frequent application daily the condition improves rapidly, but for permanent results their use must be long continued.

III.—PITYRIASIS ROSEA [1]

SYNONYMS: *Pityriasis maculata et circinata* (Bazin), *Herpes tonsurans maculosus* (Hebra).

Pityriasis rosea is a disease of the skin characterized by the development of symmetrically distributed macules or patches which are roundish or circinate in outline, slightly scaly, and of a faint red color.

The disease was described and given the name by which it is now usually known by Gibert in 1760.

Symptomatology.—The eruption occurs in two forms, the macular and the circinate. Probably the first lesion in both forms is a very small pinkish macule or papule which rapidly spreads to form the characteristic slightly scaly macule or patch of the disease.

In the macular type the lesions are roundish patches, gradually fading into the healthy skin, of a pinkish color and covered with fine branny scales. These patches vary in size from one sixteenth to three quarters of an inch in diameter, and are of a reddish to fawn color. Frequently the periphery of the lesions will show the erythematous color while the center will be fading.

The circinate type of the disease differs from the macular in that the tendency of the patches to spread peripherally is more marked. In this form the border spreads while the center fades. The typical lesion of this form, therefore, is a more or less circular patch with a pinkish border and a fawn-colored scaly center. Adjacent patches of this sort may coalesce, and thus form gyrate figures. These patches do not usually become larger than an inch in diameter. By the time they reach that size the reddish border breaks up and fades out, leaving the characteristic fawn-colored stains of the disease. The lesions are apt to occur in successive crops, so that it is possible to see at one time the minute papules at the beginning of the disease, along with the patches and circinate figures of a later stage of the process. At times the eruption remains papular for the most part throughout its entire course, only a few patches or circinate figures developing.

There is considerable variation in different cases in the color of the which at times show an inflammatory red, but more frequently a or fawn color.

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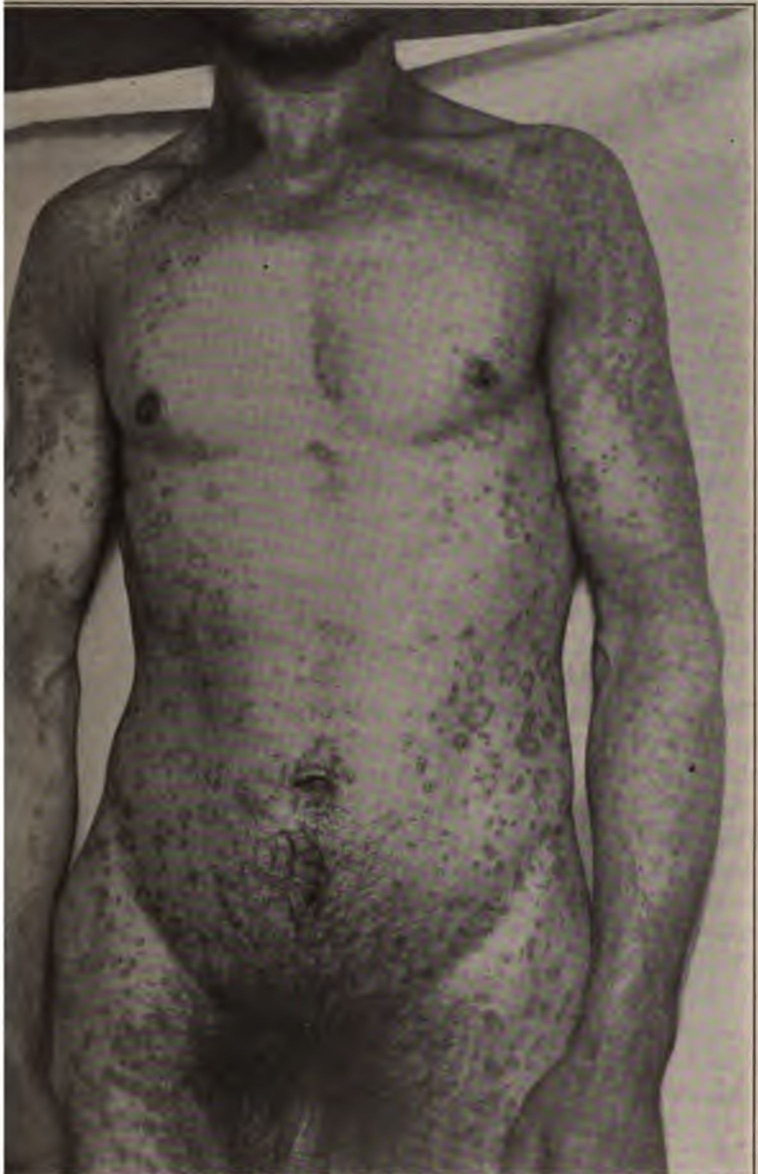
[1] Gibert, "Traité pratique des maladies de la péau," Paris, 1760.—Duhring, *Amer. Jour. Med. Sci.*, October, 1880.—Behrend, *Berlin klin. Wochenschr.*, 1881.—Vidal, *Annales*, 1882.—Fox, Colcott, *Lancet*, September 20, 1884.—Brocq, *Annales*, 1887.—Besnier, *ibid.*, 1889.—Zeisler, *Jour. Cut. and Gen.-Urin. Dis.*, 1893.—Fordyce, *ibid.*—Fox, G. H., *ibid.*—Towle, *Jour. Cut. Dis.*, April, 1904 (Analysis of 202 cases).

cq called attention to the fact that a single primary patch, usually size and situated upon the abdomen, preceded by ten days or two



99.—PITYRIASIS ROSEA, ANNULAR TYPE; macular lesions also numerous. (Author's collection.)

weeks the general eruption, and this observation has been confirmed other dermatologists.

The extent of the eruption varies very greatly. In some cases it confined to one or two regions, but most frequently it is widely distributed. It occurs by preference on the front and sides of the abdomen, over the chest and on the buttocks, and on the sides of the neck. I may be universal, but it rarely develops on the face or below the elbows and knees. The eruption usually disappears in from two or three weeks to two or three months, but cases not infrequently last longer, even for four or six months. Recurrence of the disease is rare, but occasionally happens.

The appearance of the eruption is usually preceded by a slight rise of temperature with malaise, but these systemic manifestations are as a rule so slight as to pass almost unnoticed. There may be some enlargement of the lymphatic glands. This may be confined to the submaxillary and post-cervical glands, though at times the axillary and inguinal glands also show slight painless enlargement. There is slight itching, particularly at night, or when the patient gets overheated, but it is not of severe character and causes little or no annoyance.

Etiology and Pathology.—Pityriasis rosea is one of the rarer dermatoses. It is most frequent in early adult life and in women, but occurs at all ages, in both sexes and in all classes. It is most frequent in autumn. Multiple cases in a family have been reported by Crocker, Zeisler, Fordyce, G. H. Fox, and there is a possibility that it is very mildly contagious.

Its actual cause is unknown. Hebra, as his name for it indicates, regarded it as a form of tinea, and it is probable that a form of ringworm occurs occasionally, in Vienna at least, which is indistinguishable clinically from pityriasis rosea, but it is definitely established that the usual type of pityriasis rosea is not due to any of the common mycelial organisms. Vidal thought he had found a pathogenic organism of pityriasis rosea, but later observations have failed to discover any definite parasite. As Besnier has pointed out, clinically the cases closely resemble some of the generalized cases of seborrhœic dermatitis. The symmetrical distribution, the slight febrile disturbance, and the character of the eruption suggest strongly that it is closely allied to erythema multiforme; *erythema maculatum et circinatum* would accurately describe it. There is little doubt that it is due to a constitutional disturbance.

Microscopically the changes found are those of a slight inflammatory process in the skin.

Diagnosis.—The exceedingly trivial character of the systemic disturbances and the duration of the eruption serve to distinguish it from the exanthemata. From psoriasis and widely distributed forms of seborrhœic dermatitis occurring in small patches it is distinguished by the lack of infiltration of the patches, the much lower grade of inflammation, and the character of the scales, the fine, branny scales of pityriasis rosea presenting no similarity to the coarser scales of psoriasis and seborrhœic dermatitis. From tinea circinata it is distinguished by its much wider distribution, the rapid development of the lesions, and the absence of the ringworm fungus. It is most likely to be confused with the squamous and circinate syphilides, from which it is distinguished by more rapid development, less scaling, absence from the face and hands, and absence of concomitant symptoms of secondary syphilis.

Treatment.—The disease requires very little treatment. Constitutional treatment should be symptomatic. Certain authorities think they have seen benefit from the use of salicylic-acid compounds. A mild antipruritic dusting powder, or lotion or salve, is all that is necessary in the way of local treatment. D. W. Montgomery (*Jour. Cutan. Dis.*, April, 1906) highly recommends Jamieson's treatment of pityriasis rosea, published by Walker, as follows: The patient is given daily, for half an hour, a bath containing two or three teaspoonfuls of Condy's fluid, after which the skin is anointed with vaseline containing three to five per cent of salicylic acid.

IV.—PSORIASIS [1]

SYNONYMS: *Lepra, Lepra alphos, Alphos, Psora.*

Psoriasis is a chronic inflammatory disease of the skin characterized by the occurrence of sharply circumscribed, roundish patches of all sizes up to several inches in diameter, which have an infiltrated red base and are covered by dry, whitish, adherent scales.

Symptomatology.—The elementary lesions of psoriasis are small, reddish papules, slightly elevated and infiltrated, and capped with whitish adherent scales. These lesions slowly enlarge at the border formation of round, sharply circumscribed scaly patches, w exactly the same characteristics as the primary papules. T

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[1] Ries, *Archiv*, vol. xv, 1888 (review of subject with bibliography).—Nielsen, *Monatshefte*, vol. xv, 1892 (full review); *New Syd. Soc.*, "Sel. Monographs on Dermatology," 1893.—Robinson, *New York Med. Jour.*, July, 1878.—Jamieson, *Edinburgh Med. Jour.*, January, 1879.—Pye-Smith, *Guy's Hospt. Reports*, vol. xxv, 1880–81.—Thin, *Brit. Med. Jour.*, September 4, 1880; July 30, 1881.—White, J. C., *Amer. Jour. Med. Sci.*, January, 1885.—Greenough, *Boston Med. and Surg. Jour.*, September 10, 1885.—Hebra, Jr., *Monatshefte*, vol. vi, 1887.—Schutz, *Archiv*, vol. xxiv, 1892.—Bulkley, *Trans. Med. Soc. of State of N. Y.*, 1895 (Treatment).—Rille, *Wien. med. Wochenschr.*, 1895 (In Children, Bibliography); *Dermatolog. Zeitschrift*, November, 1898.—Hallopeau et Gasne, *Bull. de Soc. française*, July, 1898.—Munro, *Annales*, 1898.—Kopytowski, *ibid.*, 1899.—Bosellini, *Monatshefte*, vol. xxix, 1899.—Hartzell, *Amer. Jour. Med. Sci.*, September, 1899 (Bibliography).—Dreuw, *Monatshefte*, vol. xxxiv, 1903; *Jour. A. M. A.*, June 10, 1905.

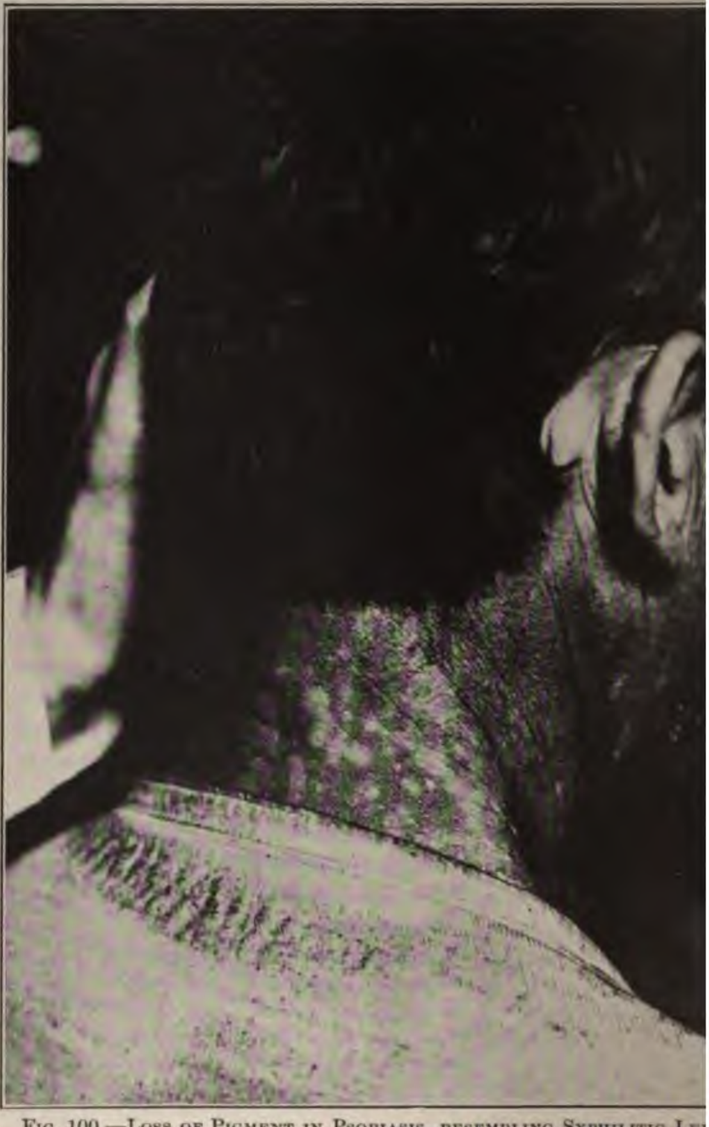

FIG. 100.—LOSS OF PIGMENT IN PSORIASIS, RESEMBLING SYPHILITIC LE
(Author's collection.)

lesions are all formed from the enlargement of these individu and not from the coalescence of elementary papules. Pso disease which presents only one type of lesions: lesions whi

ory base and a scaling surface, never becoming vesicular
lar, and never, except from secondary changes, showing

in the acute exacerbations of psoriasis, the process is of
ntensity; the base of the lesions is of red or dull red color, is
filtrated, very slightly elevated, and has a sharply circum-
nded border. The border extends for a small fraction of an
d the scales which cap the lesions, and shows as a reddish
is base does not differ from that of any ordinary subacute
ry process. The scales of psoriasis are characteristic. They
mbricated, horny scales, in typical lesions silvery or grayish
en they are scant they form a thin, laminated layer; when
they may be heaped up into a thick, grayish-white, dry crust,
s like a layer of dry mortar on the surface. From acci-
tures their typical character may be somewhat altered.
be made more or less brownish or blackish looking from
ith dirt. In individuals who perspire freely, or on sur-
re there is an abundance of sebaceous secretion, they may
llowish and slightly greasy. On the legs or elsewhere, where
are heaped up into thick crusts, there may be slight moisture
base of the lesions, gluing the crust into a softish yellow mass,
rule the scales are dry and hard, and preserve their peculiar
ppearance. The outermost scales may be loose and easily rubbed
he deeper scales are closely adherent to the base of the lesions,
be detached only by exercising force. Upon their removal the
ws as a red, glistening epidermal membrane, with almost micro-
ddish dots, representing the tips of the papillæ and, what can
readily seen, minute points of hæmorrhage where the tips of the
have been torn off in removing the scales. The scales of psoria-
relatively abundant. There is hardly any other disease in which
ick masses of true scales occur as in the extremely scaly lesions
iasis. The quantity of the scales varies in a general way with
of the lesions; over a single papule the scaling will consist of
few scales which whiten the tip; in coin-sized lesions the surface
ually be covered with a palpable layer of white scales sufficiently
o obscure entirely the underlying red base. In patches of psoriasis
size of a hand or larger, the scales may be heaped up into masses
rter of an inch or more in thickness. Cases vary in the amount
ling which occurs, even regardless of the size of the lesions, but
important than this individual variation in the quantity of the
is the variation produced by the conditions under which the lesions
. Where they are frequently washed with soap and water, or where
iration is free, or where they are exposed to constant irritation,

the scales may form a very thin layer over the base or leave the infla matory base exposed without evidence of scaling.

The skin in the patches of psoriasis becomes inelastic from infilt tion, and so upon parts where movement is extreme fissures of mech

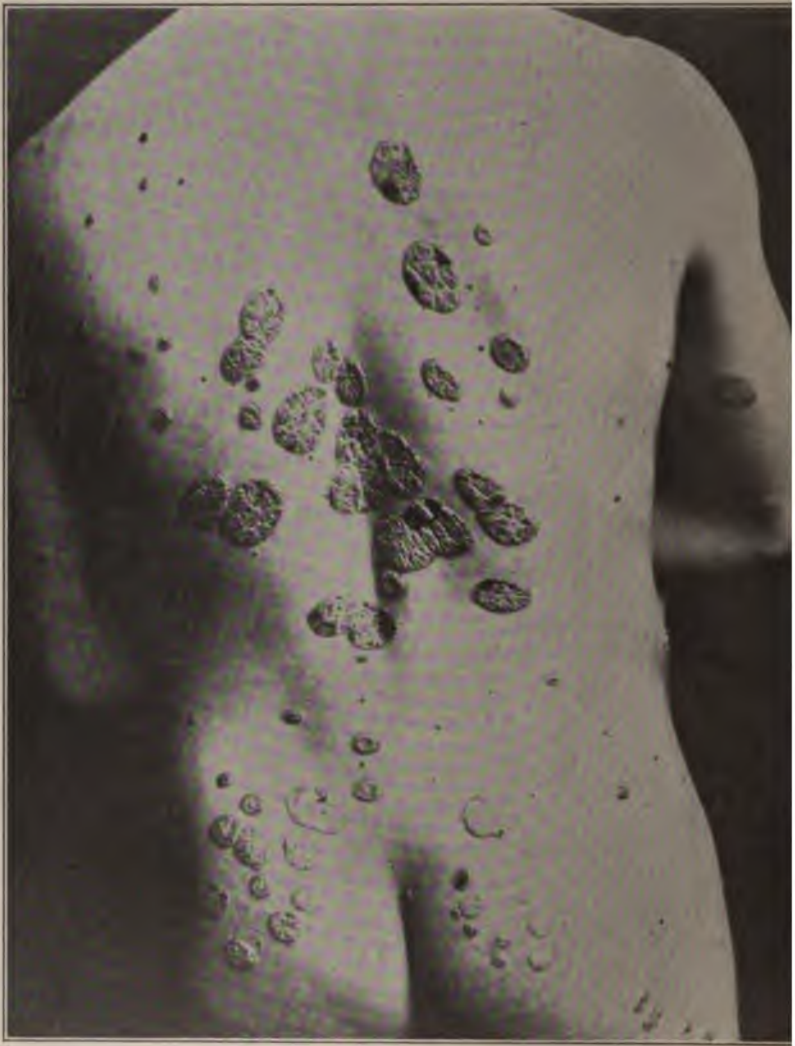

FIG. 101.—PSORIASIS—NUMMULAR TYPE, WITH ABUNDANT SCALING. (Grover W. Wende's collection.)

ical origin may be produced; but these are not common except extreme cases. In cases that are of rather acute intensity it is infrequent to see rounded or irregular excoriations in the patches f the forcible removal of the scales in scratching. Aside from these accidental features, the bases of the lesions show a remarkable uniformity in their appearance.

Psoriasis patches, after reaching a certain size, are apt to remain stationary. According to the size which the patches attain, considerable variety is seen in the appearance of the eruption as a whole. To these various clinical pictures of psoriasis different descriptive names are given, and they are convenient but indicate no essential differences in the types. The disease may remain papular throughout its course—*psoriasis punctata*—or the lesions may increase to the size of a drop or larger, and dot the surface with round, scaly patches not more than a quarter or three-eighths of an inch in diameter—*psoriasis guttata*; or the patches may reach the size of a coin—*psoriasis nummularis.* Individual lesions not infrequently reach a much larger size, covering an area several inches in diameter. More frequently the large patches of psoriasis are formed by the coalescence of adjacent patches. The largest lesions of this sort are most frequently seen over the lumbar spine, where they may coalesce into a plaque that covers most of the lower part of the back.

The involution of psoriasis patches occurs at the center. In the coin-sized and smaller lesions the process is usually diffuse throughout the lesion, and central involution is not apparent, or requires careful examination to determine it. Even in coin-sized lesions, however, the process may be more active at the border than in the center. In the larger lesions it is not infrequent to see an active border with a subsiding or normal center, thus forming ringed lesions—*psoriasis circinata seu annulata.* When these ringed lesions coalesce, gyrate or polycyclic figures are formed—*psoriasis gyrata.* In this way striking, large gyrate figures are frequently produced.

Psoriasis has a peculiarly characteristic distribution. The disease usually begins on the extensor surfaces just below the knees and elbows, and rarely exists without the occurrence of lesions in these locations. With almost equal regularity the scalp is involved. Other areas of predilection are the trunk, especially the lower part of the back, and the extensor surfaces of the limbs. The flexures are rarely involved. From the scalp the lesions may spread down somewhat beyond the hairy border upon the forehead, forward beyond the hair line behind the ears, and downward on the neck. Beyond this the face and neck are not often involved, although in abundant eruptions lesions on the face occasionally occur. The hands and feet also are rarely involved. In very exceptional cases it occurs on the palms, but never without lesions on other parts. In very rare instances the characteristic distribution is departed from. It is not uncommon, in connection with the disease

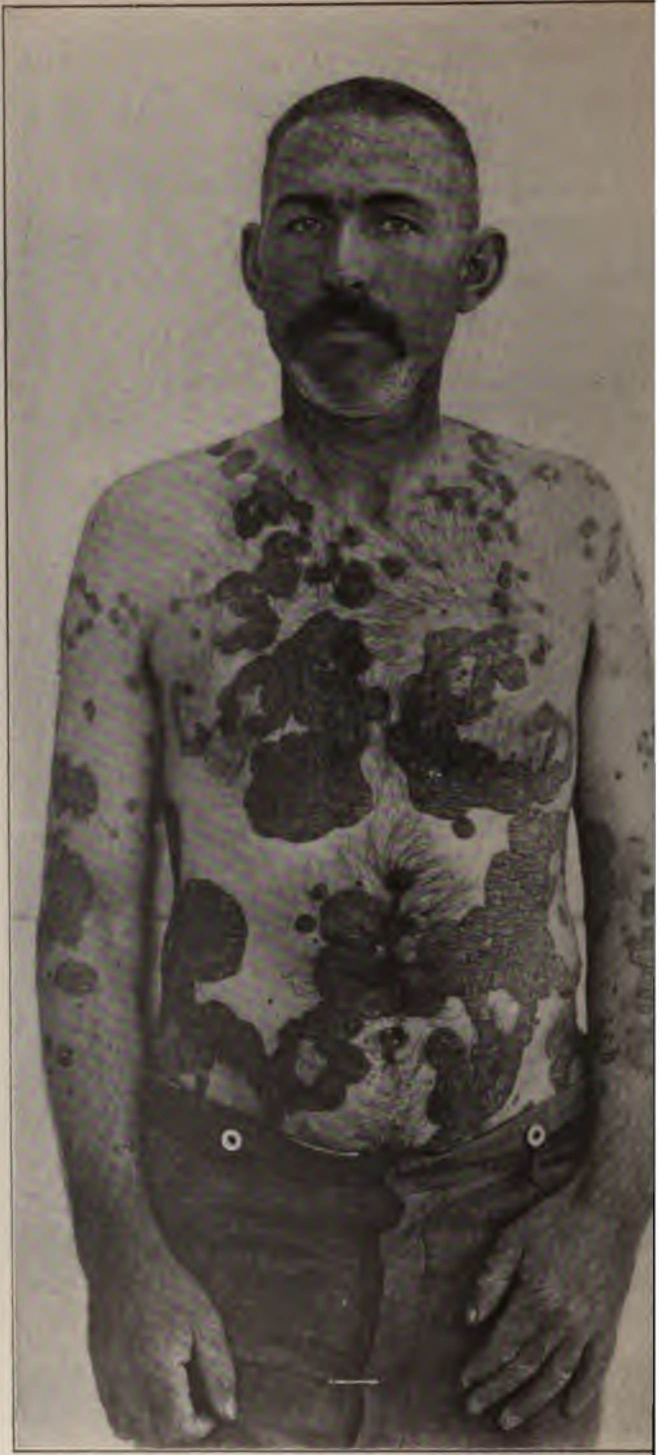

FIG. 102.—PSORIASIS. Gyrate figures from cralesence of patches. Base of deeper red than usual, scaling moderate. (Author's collectio

elsewhere, to see lesions develop at sites of irritation or injury, or in scars, as on tattoo marks and vaccination scars, or in scratch marks. Thibierge, Hallopeau, and Gasne have noted cases in which the disease apparently followed nerve distribution, or, to refer to what is probably the more essential factor, the lines of cleavage of the skin. The disease does not involve the mucous membranes. To this statement the excep-

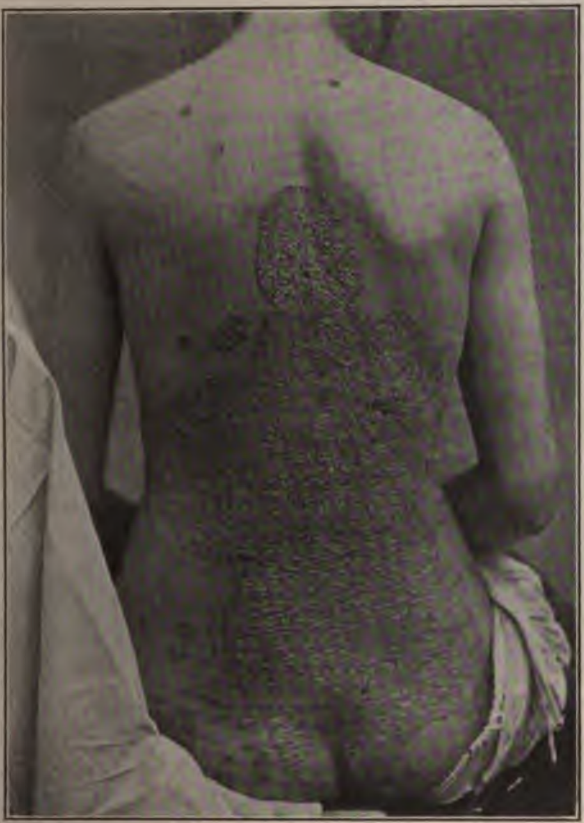

FIG. 103.—PSORIASIS IN LARGE DIFFUSE PATCHES. (Schamberg's collection.)

n must be made that Kuznitzky and Sack have reported two cases which lesions appeared on the mucous membranes. On the scalp the ease is almost invariably associated with abundant scaling, which ms thick, laminated masses. Usually these show the dry, harsh, itish character, but occasionally they are greasy and softish. The tches on the scalp may be sharply defined, coin-sized, roundish patches, parated by areas of uninvolved skin. In extreme cases they may cover e entire scalp with a thick, confluent cap of scales. Unlike seborrhœic rmatitis, the growth of the hair is slightly or not at all interfered with by psoriasis. The hair is usually dry and harsh, but is not, as a rule, appreciably thinned by the disease.

The appearance of lesions around and under the nails may cause their involvement. They become brittle, thickened, whitish, and opaque, and the free borders may be uplifted. The changes in the nails produced by psoriasis are indistinguishable from those produced by other subacute inflammatory processes.

The eruption of psoriasis may be limited to a few lesions about the elbows and knees and on the scalp. The disease may continue thus for weeks and months, and then develop suddenly by the irregular appe ance of lesions into an abundant eruption upon various parts of t body. Frequently it begins not simply on the elbows and knees, b at the same time upon other parts of the body. As a rule, in psoria the lesions are numerous over the trunk and extensor surfaces, and extreme cases a large part of the body surface may be involved. Eve however, in extensive cases, the lesions are for the most part discre In rare cases the disease has been observed to spread over the enti surface and develop into a condition indistinguishable from gener exfoliative dermatitis.

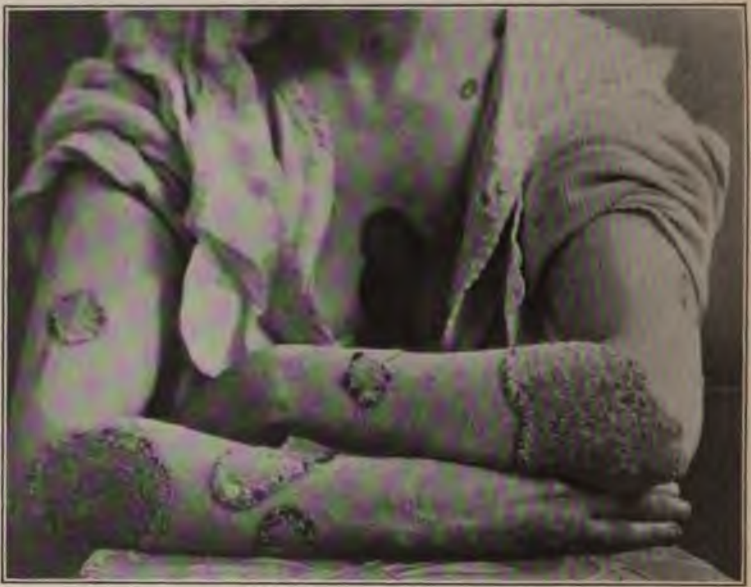

FIG. 104.—PSORIASIS IN ANNULAR PATCHES, WITH A TENDENCY TO CENTRAL INVO TION, AND WITH MORE THAN USUAL SCALINESS AT THE BORDERS. (Winfie collection.)

Psoriasis is essentially chronic and capricious in its course. T eruption appears, perhaps continues for several months, it may be var ing greatly or little in intensity; then it may entirely disappear. After disappearance it may relapse at any time, and sooner or later recurrence practically always occurs, the intervals between attacks varying from a few weeks to several months, not often more than a year. The eruption usually develops slowly, and becomes extensive gradually. Lesions appear without any regularity, and pursue an equally irregular course. Patches may persist for months or indefinitely; on the other hand, they may spontaneously disappear. Occasionally the disease appears rapidly, and becomes widely generalized in a short time. These cases are usually at first of an acute inflammatory type, but later they are apt to assume the ordinary subacute type of the disease and pursue its chronic course.

Upon undergoing involution the patches disappear without scarring. First the scaling becomes less and the bases less infiltrated, and finally there is left a pink spot, which quickly fades out, leaving no trace. Below the knees there may be some pigmentation. Crocker and McCall Anderson have reported anomalous cases followed by scarring, and Hallopeau, Unna, Caspary, and others loss of pigment at the sites of the patches.

Kaposi, Waelsch, and Besnier have noted the development of papillary warty growths in psoriasis patches on the extremities and on the hands. A serious complication of psoriasis which has been noted not infrequently is the development of epitheliomata in old psoriatics. Hartzell, who has reviewed these cases, is inclined to attribute the development of epitheliomata to the long-continued use of arsenic. In a case of mine, illustrated under Epithelioma, in which an enormous epithelioma developed upon the back at the site of an old patch of psoriasis, the patient had never had extended treatment, and had never used arsenic, so far as she knew, certainly never for any length of time.

The subjective symptoms of psoriasis are slight. Where the lesions are small, even though abundant, there are usually no subjective symptoms. When the skin is extensively involved there is some feeling of tension and stiffness, and some itching. Occasionally this is sufficient to cause annoyance. When the skin is involved in large areas and considerably infiltrated, there may be fissures which are painful. But usually the subjective symptoms are unimportant except in acute cases. In the not infrequent cases in which psoriasis for a time takes on a more acute type the itching may be considerable, but it is never of the intense character seen in eczema and severely itching dermatoses.

Etiology and Pathology.—Psoriasis is one of the commonest of the specific inflammatory dermatoses. It constitutes two to three per cent of skin diseases, and is a disease frequently seen by the general practitioner. Concerning its essential cause we know nothing. It occurs as frequently in one class of society as another. It is a little more frequent in males than in females. It usually first appears during adolescence or early adult life. Its first appearance is relatively infrequent after forty or before eight years. In young children it is not common, but it is occasionally seen in infants. The youngest reported appearance (Rille's case) is at six days old. It is usually better in summer than in winter, but in its actual causation climate, so far as we know, has no effect. Heredity is a factor in a good many cases (thirty per cent, Erasmus Wilson). It is not uncommon to find it in parent and child, or in collateral members of a family; on the other hand, it is not at all common to find it in brothers and sisters, and there is nothing in its occurrence to suggest that it is in the slightest degree contagious.

As to the influence of the general health upon the causation of psoriasis, there is a difference of opinion. A lowered condition of the general health, physical and mental overwork, digestive and nutritional disturbances, pregnancy, alcoholism and other excesses, are sometimes regarded as exciting causes. In individuals who have previously had attacks of the disease such influences may excite it, and they undoubtedly frequently intensify the disease when present, but that these disturbances of the general health ever are actual causes of psoriasis is extremely doubtful. Indeed, a striking fact with many psoriatic patients is their vigorous health, as was long ago pointed out by Hebra. The chief exception to this in my experience is the not infrequent association, in a way that seems essential, of psoriasis and rheumatic and gouty tendencies.

The pathological anatomy of the lesions of psoriasis may be summarized as vascular dilatation, œdema and slight cellular infiltration of the corium, and moderate œdema of the epidermis with anomalous cornification, hyperkeratosis, and proliferation of the interpapillary processes. The majority of the German observers consider the inflammatory changes in the papillary layer of the corium to be primary; Unna thinks the first lesion is parakeratosis; Robinson and many English writers believe the primary lesion to be hyperplasia of the epithelium; while Crocker, Brooke, and others consider the changes in the corium and epidermis to be synchronous, and due to the same cause acting on both simultaneously.

The vessels of the corium, especially of the papillary layer, are dilated and congested, and there is a cellular infiltration, most marked around the vessels and glands, composed of polynuclear and mononuclear cells. There is some proliferation of the endothelial and connective-tissue cells. The papillæ are enlarged in both length and breadth. In the epidermis there is marked proliferation of the interpapillary portions of the rete mucosum, causing enlargement, laterally and downward, of the interpapillary processes; over the papillæ the epidermis is not much thickened, a fact which explains the punctate hæmorrhage when the scales over the dilated papillary vessels are removed. There is slight intercellular œdema. The transitional layer is absent, as are keratohyalin and eleidin granules; hence the vivid red color of the scaleless papule. The horny layer is much thickened by reason of persistence of the cells, which are moist, retain their nuclei, and are deficient in keratin—parakeratosis. When the cells become partially dried, air penetrates between them, producing the silvery-white luster of the scales. The cells, instead of rubbing off singly, adhere together, producing lamellæ, which are often separated by collections of leucocytes. Several observers claim that these microscopic foci of leucocytes are the primary lesions of the disease.

While many investigators have found microörganisms in the lesions of psoriasis, its microbic genesis and communicability are unsettled questions. Unna attributes the disease to the growth in the epidermis of his morococcus. Destot has successfully inoculated himself from a case of vaccinal psoriasis, and his disease recurs every spring. Unna, Meneau, Graves, Cantrell, Porr, Nielsen, Anderson, Aubert, Hammer, and others have reported cases of probable contagion. On the other hand, Ducrey in numerous experiments has entirely failed to reproduce the disease by inoculation. Lang's epidermophyton has been shown to be eleidin granules. Oro and Mosca were also unable to obtain positive results from inoculations, but found on autopsy, as have others, myelitis and interstitial neuritis. Crocker believes that the disease is due to a microörganism, and Hallopeau also advocates this theory; the majority of authorities regard it as improbable.

Diagnosis.—The diagnostic features of psoriasis are the sharply defined, rounded lesions, which have a chronic inflammatory base and are covered with white, dry, adherent scales, the distribution, and the persistent tendency to recur. Well-defined cases can hardly be confused with any other dermatosis. The eruptions with which it is most likely to be confused are scaling papular syphilides, seborrhœic dermatitis, and scaling eczemas.

Papulo-squamous syphilides may resemble guttate lesions of psoriasis very closely, but they are always associated with other evidences of secondary syphilis, like mucous patches and condylomata, and there is usually no itching. Their scales are dirty and brownish, rarely white and shiny, and the bases of the lesions are of a dull red or ham color. Further, the syphilides avoid the areas of predilection of psoriasis, and are likely to occur on the face, palms, and soles. The scaling tubercular syphilides of tertiary syphilis are of darker color, show less scaling, are associated with ulcerative lesions, atrophic scarring, and pigmentation, are of irregular and unsymmetrical distribution, and occur frequently on the face.

Patches of squamous eczema which superficially resemble patches of psoriasis have less scaling, are of a fainter or a brighter red color, have ill-defined borders which gradually fade out into the healthy skin, and in or around the borders inflammatory papules or vesicles are likely to be found. Patches of eczema are of irregular distribution, occur by preference upon flexor surfaces, frequently involve the hands and face, and are likely at some stage of their course to show vesicles or weeping areas.

The superficial resemblances to psoriasis which such diseases as lichen planus, pityriasis rosea, ringworm, and lupus erythematosus may present at times are easily cleared up upon careful examination and consideration of the common characteristic features of these different diseases.

Prognosis.—As a rule it is not difficult to clear away an outbreak of psoriasis, but no assurance can ever be given against the recurrence of the disease. The eruption may recur almost immediately upon its removal, or it may not recur for months or a year or more. The ease with which an attack can be cured varies, as a rule, directly with the age of the disease and the age of the attack. The earlier treatment is begun after the appearance of the eruption the more readily is it removed. In the earlier attacks treatment is nearly always successful in a comparatively short time. When the disease becomes widely generalized and much infiltrated, as happens in long-standing cases, the treatment becomes longer and more difficult.

Treatment.—If a psoriatic patient is in reduced health, every effort, of course, should be made to restore him to normal, but in the ordinary cases of psoriasis we get little encouragement from meeting general indications. The patients are either in normal health or are far from relieved of their eruptions by the rational treatment of any dyscrasiæ which exist. As in eczemas of constitutional origin, we see many patients with psoriasis who are of full habit, gouty, or rheumatic, or who show some other evidence of autointoxication. In such cases a certain amount of benefit is gained by the treatment of these conditions, and alkalies, diuretics, the salicylates, and occasionally colchicum are indicated in various cases. Further than this, perhaps something can be done by improving the habits of some individuals. The diet should be regulated and exercise advised according to general indications. Where the patients are excessive users of alcohol, tobacco, coffee, or tea it is probable, in the very nature of things, that these do some harm. But all these measures for the regulation of the general health, it must be confessed, are chiefly advisable because they are proper in themselves, and not because they have any very marked effect upon psoriasis. In intervals between attacks they may stave off an outbreak for a longer or shorter time, and the probability of that being the case is sufficient to warrant advising them.

Internal Treatment.—The real treatment of psoriasis must be directed to the eruption. It is to be carried out partly by the use of certain specific internal remedies which have empirically proved valuable, but chiefly by the use of stimulating local applications. Various remedies have at one time and another been advocated for their specific effect in psoriasis. Of these, arsenic is the most certain. Others are potassium iodide, the salicylates, salicin, oil of turpentine, oil of copaiba, tincture of cantharides, tar, carbolic acid, wine of antimony, thyroid extract, pilocarpin.

As in most other chronic inflammatory processes of the skin, arsenic is useful in the chronic inflammatory type of psoriasis; it is contraindicated in the acute inflammatory cases. With arsenic, psoriasis may frequently be entirely cleared up without the use of local applications. Under its continuous administration the disease may be held in abeyance for a longer or shorter time, but is likely sooner or later to recur, and arsenic should not be given over indefinite periods in psoriasis in order to prevent recurrence of the disease, because of the danger of arsenical keratoses and of other troubles from its persistent use. It is most useful in the beginning of attacks; in old cases that have been much treated with arsenic its value is very slight. In using arsenic, it should be carried up to the point of producing its first evidence of physiological effects, then reduced below this, and continued until about a month after the disappearance of the eruption. It may be administered in any of the usual forms. Solution of potassium arsenite—Fowler's solution—is usually given, beginning with a dose of 3 gtt. t.i.d., and increased 1 gtt. daily until 8 gtt. or 10 gtt. t.i.d. are taken, or until puffiness about the eyelids or gastrointestinal disturbances appear. With Fowler's solution as with the other arsenical preparations, if benefit is not seen from moderate doses the chances are that none will be obtained by pushing it. A less commonly used preparation of arsenic which I have found satisfactory is sodium cacodylate in doses of ½ grain to 1½ grs. t.i.d. It promptly gives a garlicky odor to the breath, but does not readily disturb the digestion.

A much more prompt effect is gotten from arsenic by its administration hypodermically, and when its use is strongly indicated, this is the method of preference. Fowler's solution may be used hypodermically, beginning with 3 minims diluted in four or five parts of sterile water, and increasing a drop daily. Its hypodermic administration is somewhat painful. Solution of sodium cacodylate, as obtained in commerce sterile in glass capsules, can be used hypodermically without irritation, and it is the most eligible form for hypodermic use. It may be given in doses of ¾ grain to 2½ grains daily. The administration of arsenic in psoriasis sometimes causes temporary pigmentation at the site of the patches.

The use of large doses of potassium iodide, as recommended by Haslund, and of other alkalies for their specific effect, need only to be mentioned. In doses of ordinary size they are certainly not curative, and probably have only such value as comes from the general indications which they meet. Oil of turpentine is recommended by Crocker, administered in the same way as in eczema (*q. v.*). Oil of copaiba, tincture of cantharides, tar, and carbolic acid are sometimes beneficial, and may be tried if other things fail. The same statement applies to pilocarpin and thyroid extract, with the additional reservation that they must be used with caution, and only with the patient under constant supervision.

In the unusual acute cases of psoriasis, wine of antimony may be useful at times, as it is in other acute inflammatory dermatoses. The dose is 5 to 10 minims t.i.d., or smaller doses more frequently.

Local Treatment.—Aside from those cases in which arsenic internally is useful, successful results in psoriasis depend upon local treatment. The first indication is to get rid of the accumulation of scales, in order that the remedies to be used may be effective. For this purpose the most useful measure is daily warm bathing for ten to thirty minutes, with the free use of soap, preferably tincture of green soap. The scales are more readily dissolved if the baths are rendered alkaline by the addition of bicarbonate of soda, borax, or ammonium chloride, 2 to 6 ounces to the 30-gallon bath. After each bath the body should be greased with a nonirritating ointment, partly to soften the remaining scales and partly to overcome the dryness of the skin. The thick masses of scales which are not removed by bathing may be gotten rid of by covering them with a close-fitting layer of rubber cloth so that they become macerated with perspiration. They can also be dissolved by applications of salicylic acid, from 20 to 40 grains to the ounce of vaseline.

Upon removal of the scales we begin the radical treatment of the lesions. All of the applications used in the radical local treatment of psoriasis are stimulating, and depend for their results upon the active inflammation which they excite, although in acute cases it is needless to say soothing ointments, such as are used in other inflammatory processes, should be used until the eruption becomes of the typical subacute type. Almost any of the usual reducing and stimulating agents may be employed in psoriasis. The number, however, which experience has shown to be especially useful is small. Of these, chrysarobin bears place; then, in the order of their usefulness, may be mentioned ammoniated mercury, especially for the scalp, salicylic acid, beta- thol, and sulphur.

hrysarobin is the most effective local application, but it is difficult se. It produces more or less dermatitis upon the healthy skin, which, ever, is temporary, and yields readily to soothing applications; it ns the clothing a mahogany brown; it cannot be used on the face use of the danger of producing conjunctivitis, or on the scalp be- e it colors the hair yellow. It also temporarily stains the healthy a walnut brown, but it curiously leaves the sites of the lesions tained. Where extensively and vigorously applied in the form of ointment, it may produce a universal dermatitis, but this promptly ides, and with it the psoriasis disappears. In ointments it may be l in strengths of one to ten per cent. Zinc ointment or Lassar's paste good vehicle. The weaker preparations—two to four per cent—are n efficient, and should be tried first. The ointment should be applied e daily, and as nearly as possible only to the diseased areas, and its application the surfaces should be dusted with talcum or some r inert powder. An ointment recommended by Unna is as follows:

℞	Salicylic acid	3.0
	Pyrogallic acid	3.0
	Ichthyol	3.0
	Olive oil	10.0
	Lanolin	ad 100.0

reuw highly recommends the following plan of treatment:

℞	Salicylic acid	10.0
	Chrysarobin, Oleum rusci, āā	20.0
	Sapo viridis, Vaseline, āā	25.0

s is applied with a stiff brush to the affected areas daily for four to days. After the fifth or sixth day the patient takes a hot bath daily from one to three days. After each bath vaseline is rubbed into surfaces from one to three times a day. A day or two after begin- treatment it is found that the ointment remains on the normal around the patches as a black, parchmentlike crust, while over the ues there is intense scaling. This black crust loosens in a few days athing and of inunctions of vaseline. After eight or nine days the tment may be repeated, if necessary, but, as a rule, the psoriasis ppears soon after the first period of treatment. Unna and Lassar recommend the method. In spite of the strength of the chrysarobin, it it said to cause very little irritation.

In order to limit the application as far as possible to the diseased patches and to prevent staining of the clothing, it is better in private practice to apply chrysarobin in the form of a fixed dressing. The most efficient way of doing this is to make a paint of chrysarobin, 1 or 2 drams to the ounce of chloroform. Chloroform will dissolve about 40 grains of chrysarobin to the ounce, so that in this mixture there is a considerable amount of chrysarobin in suspension. This mixture is shaken up and painted upon the surfaces, and upon the evaporation of the chloroform a fine layer of the chrysarobin remains. This is then fixed by varnishing over with flexible collodion.

The same application may be made more conveniently, but not quite so effectively, by the use of a collodion varnish like the following:

℞ Chrysarobin	ʒi;
Salicylic acid	gr. x–xx;
Flexible collodion	fl℥i.

Or a traumaticin varnish may be used:

℞ Chrysarobin	ʒi;
Salicylic acid	gr. x–xx;
Liquor gutta-perchæ	fl℥i.

These mixtures are painted on the surfaces. The films are left until they begin to peel off, and new applications are then made.

Pyrogallic acid in the strength of 20 to 60 grains to the ounce may be used either in ointments or in collodion varnish as a substitute for chrysarobin. It is less irritating and stains the skin less, but it stains the clothing, is less effective than chrysarobin, and its extensive use sometimes causes toxic symptoms, so that it is not, on the whole, a satisfactory substitute.

Next to chrysarobin, tar is the most useful application. It may be used in the form of oleum cadini, oleum rusci, or pix liquida. These are used generally in ointments (zinc ointment or Lassar's paste are best), from ½ to 2 drams to the ounce, with the addition of 10 grains of salicylic acid. They may be used like chrysarobin in a mixture with flexible collodion, 1 or 2 drams to the ounce. The smell of tar and its dirty color are objections which can be overcome by using Duhring's compound tincture of coal tar. This is very efficient, and is used in ointments in the same strength as pure tar. When ointments are used they should be freely applied twice a day, and allowed to get into the underclothing so that the patches are constantly exposed to their action.

Beta-naphthol, gallacetophenone, anthrarobin, resorcin, and aristol, n the proportions of ½ to 1 dram to the ounce of ointment, and ous other applications are more or less useful in psoriasis, but they less effective than the remedies already considered.

For use on the scalp and about the head, ammoniated mercury comd with salicylic acid is the usual, and a satisfactory, application. used in the strength of ½ to 1 dram with 10 to 20 grains of salicylic to the ounce of vaseline or rose ointment. Before its application the should be cleared as fully as possible of scales by thorough shamng with soap and water; then the salve should be thoroughly applied y and the scalp shampooed at intervals of a few days.

Sulphur in the same strength as ammoniated mercury, and combined salicylic acid, is a useful application for the scalp, and may also dded at times with advantage to the usual prescriptions for the body.

None of the ordinary local methods of treatment of psoriasis are sfactory to the patients. They require persistence and patience in r application, are troublesome, mussy, and disagreeable, and in the thoroughly tire out the patients. For these reasons the successful lication of x-rays in these cases is a very valuable addition to our ms of treatment. Under moderate or light exposures, repeated from to day, or at proper intervals according to the amount of x-rays used, iasis usually disappears completely. It has never been necessary ny experience to produce any evident dermatitis, and I have used the tment on the scalp in a good many cases without ever causing any ecia. The treatment should be very cautiously given; one should his way along and avoid the production of dermatitis. The treatt is far more agreeable to the patients than any of the other forms reatment of psoriasis, and I have seen it successful in rebellious cases that had practically resisted all methods of treatment. My ession is that the intervals between attacks are very distinctly longer under other circumstances, but recurrence is still to be expected.

V.—LICHEN

The term lichen as descriptive of papular eruptions of the skin is d as the writings of Hippocrates. Upon the basis of that classical ority Willan, in the early part of the last century, grouped under erm lichen various skin diseases which had the single common cteristic of presenting an eruption of papules. This loose use of erm has been the cause of much confusion, and there are still variaffections occasionally described under the appellation of lichen h have nothing to do with the definite disease that is now known chen planus. Among these are:

Lichen simplex, a term applied to papular or vesicular eczemas.

Lichen urticatus, chronic urticaria in children.

Lichen pilaris, for keratosis pilaris.

Lichen lividus, lesions produced by hæmorrhage into the hair follicles, follicular purpura.

Lichen tropicus, miliaria rubra.

Lichen strophulosus, miliaria rubra in young children.

Lichen syphiliticus, follicular papular syphilide.

Lichen circinatus, circinate lesions of seborrhœic dermatitis of the body.

The term lichenification has been considered under general symptomatology.

None of these conditions has any essential relationship to lichen planus; indeed, the term lichen alone has no definite significance except as it is applied to lichen planus, and its use to denominate other diseases is better avoided.

The other conditions to which the term lichen is applied are lichen planus, lichen variegatus, lichen ruber acuminatus, lichen scrofulosus, and lichen pilaris seu spinulosus. There are certain analogies between lichen planus, lichen variegatus, and lichen ruber acuminatus which suggest an essential relationship, but the clinical manifestations of the three affections are so different that they can only be described as separate diseases. Lichen scrofulosus and lichen pilaris seu spinulosus probably have no essential relationship to lichen planus.

Lichen planus was rescued from the obscure group of the lichens in 1869 by Erasmus Wilson,[1] and it is the only one of these affections to which the name lichen can be applied without possibility of confusion. It is a distinct clinical entity.

LICHEN PLANUS[2]

SYNONYMS: *Lichen ruber planus, Lichen psoriasis.*

Lichen planus is a chronic inflammatory disease of the skin characterized by peculiar glistening, flattened papules of polygonal outline, and of red to purplish-red color.

The papules may be discrete in distribution or they may be aggreed into thickened violaceous, slightly scaly patches. The disease is ally of chronic type, but in rare cases pursues an acute course. It y be limited in distribution or generalized. The chronic form occur- g in limited patches is the usual type.

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[1] *Jour. Cut. Med.* London, vol. iii.

[2] Crocker, *Monatshefte*, vol. i, 1882; *Brit. Jour. Derm.*, 1900.—Engman, *Jou* *Cut. and Gen.-Urin. Dis.*, 1901; *Brit. Jour. Derm.*, 1904.—Lieberthal, *Jour. A. M. A* 1902.—Wickham, *Annales*, 1895.—Fox, Colcott, *Brit. Jour. Derm.*, 1895.—Alle *Trans. Amer. Derm. Assn.*, 1901; *Jour. Cut. and Gen.-Urin. Dis.*, 1902.—Cantr *Med. Record*, August 7, 1897.—Discussion, Crocker and others, *Brit. Jour. Der* December, 1900.—Ravogli, *Jour. Cut. Dis.*, December, 1904.

Symptomatology.—The elementary lesion of lichen planus is a culiar and characteristic angular papule of the shape of a truncated ramid. Its outline is polygonal, with the angles well defined, its

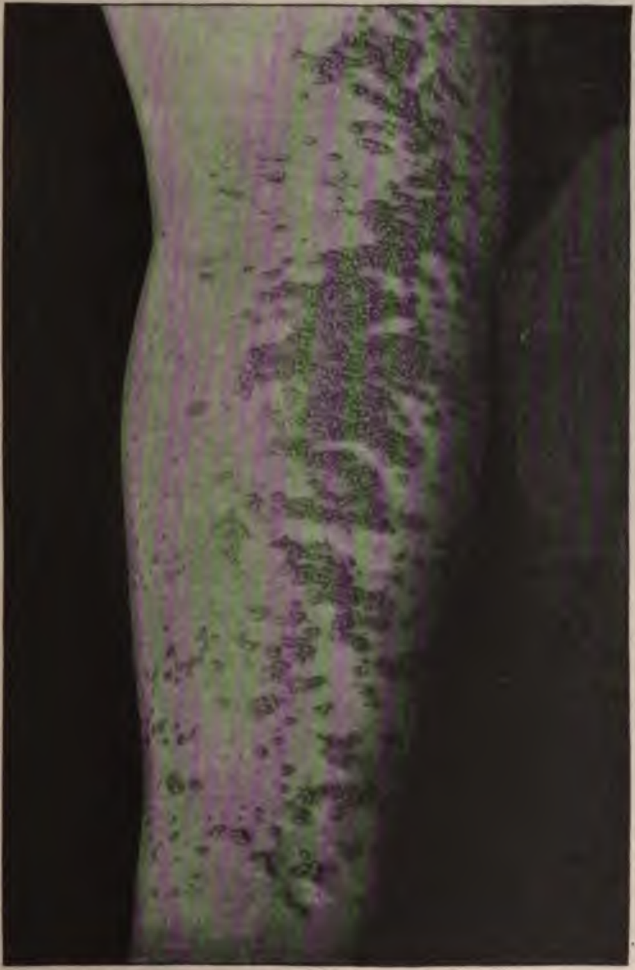

FIG. 105.—LICHEN PLANUS. (G. H. Fox, Morrow's System.)

p flat or slightly depressed, and its surface tense and glistening. he papules are sharply defined, with abruptly raised sides, and fre- uently upon close examination with a hand lens the angles of the lesions show linear prolongations like minute keloids, which give the lesions a stellate outline. The papules are usually small, somewhat larger than a pinhead, but may be a sixteenth to an eighth of an inch in diameter. The larger lesions, if of typical angular outline, usually show a central depression, which is sometimes sufficient in the largest lesions to give them an annular appearance. The color of the individual lesions is usually a dull or pale inflammatory red, perhaps sometimes violaceous; but the violaceous color is more distinctly characteristic of the patches formed from the coalescence of lesions. The lesions are tense and firm, and when looked at from the side have a peculiar shining appearance. Usually there is no scaling of individual lesions, but occasionally there are minute thin scales. Sometimes, as pointed out by Wickham, there may be seen in lesions of larger size, on examination with a hand lens, minute grayish points or striæ or red points.

The papules of lichen planus show no tendency to peripheral enlargement. The elementary papules are the typical lesions of the disease throughout its course. Patches are a common occurrence in the disease, but they are produced by the aggregation of the elementary papules. The patches are of irregular but well-defined outline, have distinctly thickened and roughened surfaces, a peculiar purplish color, and are usually covered by a thin, grayish layer of fine scales. Around or in their borders will be found typical discrete lesions. The scales of lichen planus are scant, fine, and branlike, and are not the abundant, thick, white scales of psoriasis.

The usual form of lichen planus is an eruption of groups of discrete lesions or of patches confined to a few areas of predilection. The arrangement and distribution of the individual lesions is irregular; they may be scattered and be unaccompanied at any stage by the formation of patches, or they may be arranged in groups with a tendency to the formation of characteristic patches, or in lines or circles. On the limbs the lines are usually transverse, so that the patches show transverse markings. Occasionally their distribution is determined by the presence of excoriations, as from scratching, or of other superficial wounds, or of scars, as the scars of vaccination.

The sites of predilection of lichen planus are the flexor surfaces of the wrists and forearms, the inner sides of the knees, and the lower part of the legs. These sites are characteristic, but the eruption may occur upon any part of the trunk and extremities. It rarely affects the face, the palms, or the soles. Occasionally it involves the dorsal surface of the fingers, and may then involve the nails and produce changes such as are seen from other chronic inflammatory processes in the nails. On the legs the violaceous color of the lesions is most pronounced, the individual papules are likely to be larger, and the patches are more infiltrated and present a roughened, often distinctly warty, surface.

Lesions on the mucous membranes are not infrequently observed, and should always be sought for. They occur as inflammatory papules with a grayish surface, like points that have been touched with nitrate of silver, and do not show the characteristic angular outline of the lesions of the skin. The recognition of their character depends upon their association with lesions on the skin. Lesions have not infrequently been observed upon the glans penis and on the vulva, usually as inflammatory papules topped with grayish epithelium, at other times as typical lichen planus papules.

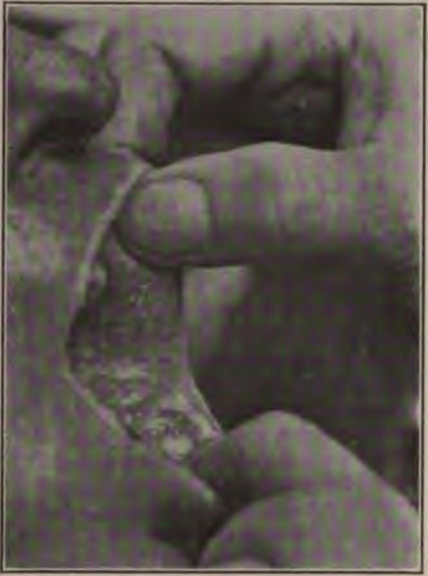

FIG. 106.—LICHEN PLANUS IN MOUTH. (Engman and Mook's collection.)

The extent of lichen planus varies between the widest extremes. It may be confined to a single group of lesions or a single patch; it most frequently occurs as one to several patches of the eruption at the sites of predilection. Occasionally the patches are numerous, and then they usually show a rough symmetry. At times, particularly in acute cases, the disease occurs as a widely generalized eruption. In such cases it is in the form of an abundant efflorescence of discrete but closely set papules showing a few areas where the papules have coalesced into patches. Even in abundant cases, healthy islands of uninvolved skin will be found. The eruption never becomes universal.

Lichen planus, as a rule, begins insidiously, and pursues a chronic course. The groups of lesions or the patches develop gradually, and after development are persistent. New lesions tend to appear, and there is some tendency to gradual spreading, but after reaching a certain size the lesions show no tendency to enlarge and little tendency to disappear. After persisting for a longer or shorter time, the eruption may disappear spontaneously. Occasionally there is a tendency to recurrence, but this is not marked as in psoriasis. On disappearing, the lesions and the patches leave light- to dark-brown stains, which fade slowly. Occasionally the disappearance of the lesions is followed by atrophic scarring.

In rare cases—in about ten per cent of the cases, according to Crocker—lichen planus occurs as an acute eruption. The acute form

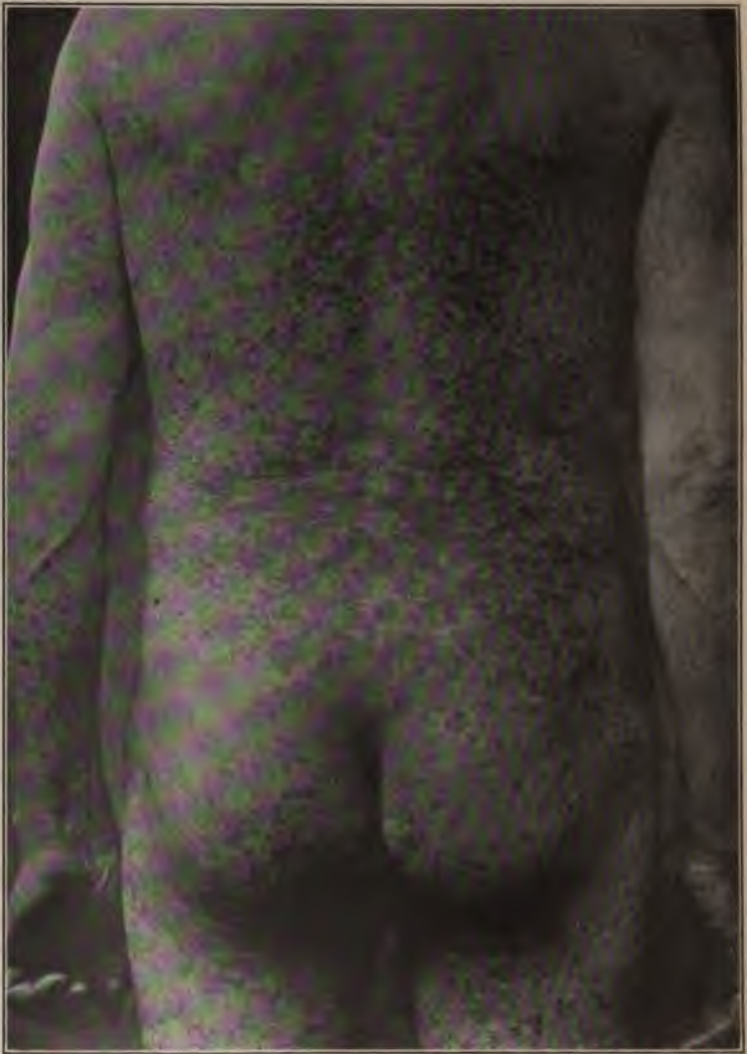

FIG. 107.—PIGMENTATION FOLLOWING LICHEN PLANUS. The stains indicate the abu dance and distribution of the eruption in a generalized case. (Engman and Mook collection.)

may either supervene upon typical lichen planus or develop *de nov* The lesions in acute lichen planus are of the same type as the ord

en planus papules, but are of small size, and show the char-
s of a more acute inflammatory process. In acute lichen planus
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sual type of lichen planus
haracteristic constitutional
ce. The acute cases may
ciated with systemic dis-
, but as a rule the patients
us, with disturbed sleep,
as the result of itching,
other evidences of consti-
depression. Severe cases
sionally accompanied by
rile disturbances, which
wed by extreme depres-
slow convalescence. In
, acute cases death even
r.

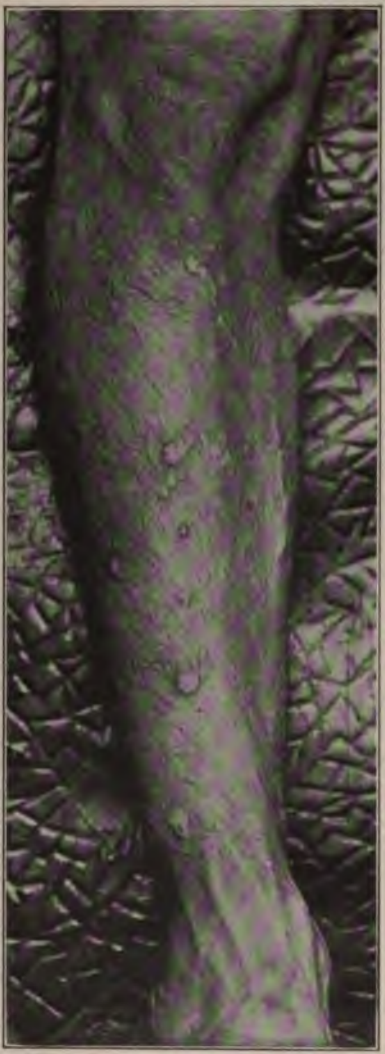

FIG. 108.—LICHEN PLANUS OBTUSUS, DISCRETE LESIONS. (Author's collection.)

tions.—At times the le-
lichen planus vary from
cal flat, angular character.
lly the lesions are convex,
nical. The convex lesions
perhaps as large as a large
n obtusus, lichen planus
hicus. Such lesions on the
sually dark brownish red
h, with slight scaling. The
e of the larger lesions of
nus, whether obtuse or flat,
when they are located on the legs, may produce elevated, thick-
gh, dark purplish, warty plateaux—*lichen planus verrucosus.*
s groups of lichen planus lesions occur in annular forms. The
rrangement may be the primary distribution of the lesions or

be produced by the formation of a circle of satellite lesions around a central older lesion which undergoes involution, leaving a ring, *lichen planus annularis.*

In rare cases, while most of the lesions are of the typical character of lichen planus, vesiculation occurs upon the summits of some of th lesions. This vesiculation is not, as has been thought, due to arseni as in nine of seventeen cases collected by Whitfield arsenic had n

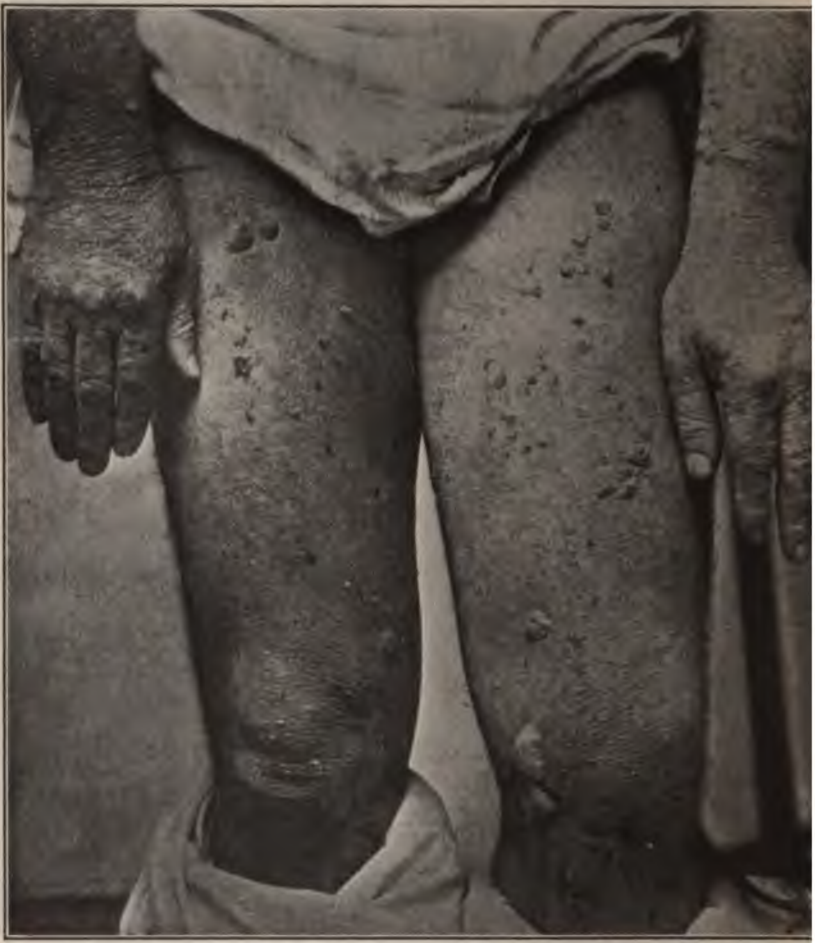

FIG. 109.—LICHEN PLANUS BULLOSUS. (Engman and Mook's collection.)

been taken. This very rare occurrence of vesicles in lichen planus the only departure of these lesions from their solid character.

Under the name *lichen ruber moniliformis,* Kaposi first describe very rare picture in which fine globular lesions were arranged in mon n bands almost like lines of beads, forming raised, scarlike linear ls on the extremities parallel to their axes. Other cases of this type have been observed by Róna, Dubreuilh, G. H. Fox, and

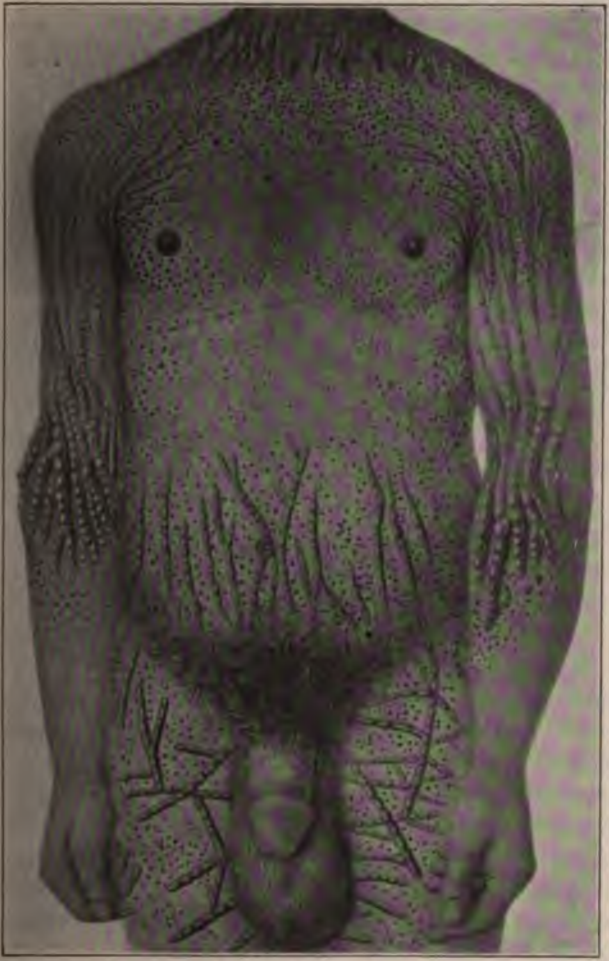

Fig. 110.—Lichen Ruber Moniliformis.
(Photographic reproduction from Neumann's colored plate.)

vsky, the lesions usually appearing symmetrically upon the ex-
ities.

n all of the above varieties of lichen planus, typical lesions of lichen us are usually associated with the anomalous forms. If these are it the diagnosis may be uncertain and depend chiefly upon the nic, persistent, unchanging character of the lesions, their distribution, and their violaceous color. In lichen ruber moniliformis ordinary lesions of lichen planus may be absent, and indeed the connection between this disease and lichen planus has been questioned. Its general characteristics, however, seem to indicate that it is a condition closely related to or essentially the same as lichen planus.

Etiology and Pathology.—Lichen planus is one of the less common affections, forming one to two per cent of skin diseases. Concerning its actual causes we have no positive knowledge.

It occurs in both sexes, distinctly more frequently in women in America and in England, but more frequently in men in Vienna. It is most frequent in the active period of adult life, from thirty to sixty, and is rare in children and in old age; Kaposi has observed it at eight months and Crocker at seventy-four years. It is, in my experience, quite as frequent among the well to do as among the poor.

In some cases there is no evident disturbance of health associated with it, but usually the patients are not in vigorous health. In a majority of cases the patients are nervous, depressed, or neurasthenic, often as a result of worry, anxiety, overwork, or some other nervous strain. This association is so common that there is little doubt that nervous disturbance is in many cases an essential factor in the causation of the disease. Sometimes the cases are associated with disturbances of digestion or of the generative organs. The disease has sometimes followed injuries. Crocker believes the acute cases are frequently determined by sudden chilling during perspiration, especially in persons who have already had the chronic form of the disease.

The microscopic anatomy of lichen planus is constant and quite characteristic. Its features are marked round and lymphoid cell infiltration of the papillary layer, without plasma or giant cells and with few leucocytes, hyperkeratosis, hyperplasia of the rete, and œdema of the epidermis. The dense papillary infiltration begins as a formation of new cells around the vessels, and is composed principally of epithelioid and fibroblastic cells, with many lymphoid cells and a few mast cells and leucocytes. Pigment cells are numerous. The vessels and lymphatics are dilated, and their walls are thickened. In the center of the papule there is frequently hyaline degeneration of the collagen. Crocker considers that the ducts of the sweat glands act as determinants for the starting of the infiltration; but it occurs also aside from them. The papillæ are in large part obliterated.

Over the cellular infiltration the corresponding epidermis is much thickened. There is hyperplasia of the rete, with intra- and extracellular œdema; the cells are greatly swollen, and show nuclear degeneration. A few leucocytes and lymphoid cells appear in the epidermis. The granular layer is thickened, especially in the center. The horny layer is thick, firm, and the cells do not contain nuclei. Over the center of the papule the hyperkeratotic horny layer forms a firm oval horny mass or plug, which pushes downward into the cells below, and later becomes loosened and falls out, causing the central umbilication of the papule.

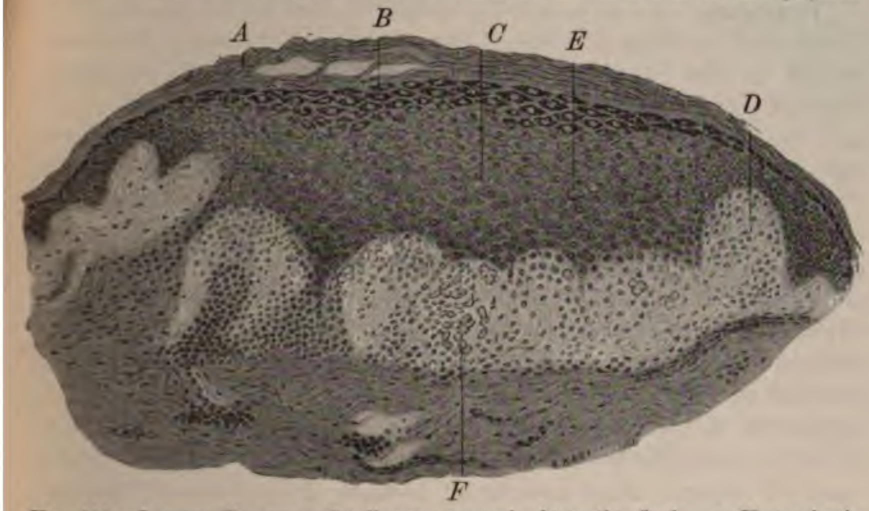

FIG. 111.—LICHEN PLANUS. Small recent papule from the flank. *a*, Hyperplastic corneum; *b*, stratum granulosum; *c*, hypertrophied stratum mucosum, composed of large cells separated by intercellular edema; *d*, edematous papilla showing infiltration with small round cells; *e*, cells showing colloid degeneration; *f*, colloid degeneration of cells in Malpighian layer. (Darier.)

Diagnosis.—The characteristic feature of lichen planus is the peculiar polygonal papule with flattened or slightly depressed top, solid, angular, of sharply defined outline, and frequently with minute ridges radiating at the angles. Examination with the hand lens will usually show a central depression or horny point, and often Wickham's grayish points and striæ. Even in atypical cases characteristic papules can nearly always be found near the borders of the patches or upon other parts of the body. Unless the typical lesions of lichen planus can be found, the diagnosis may remain in doubt. Other suggestive features of lichen planus are the purplish color of the patches, their slight grayish scaliness, their distribution, their infiltration, and their more or less roughened surface. The inexperienced may mistake for the angular papules of lichen planus the pseudo-papules which are seen on dry, infiltrated patches of dermatitis as result of the markings which are caused by the exaggerated lines of the skin. These pseudo-papules fit together, evenly and smoothly like the pieces in a mosaic; they are all alike, and at the border of the patches of dermatitis the infiltration gradually fades away without any evidence of discrete angular lesions which by their coalescence might have formed the larger patches.

Lichen planus may be confused with any of the chronic infl matory dermatoses, such as chronic eczema or psoriasis, and poss with the small papular syphilide. The crucial point in the diag is the discovery of the typical papules of lichen planus.

Prognosis.—In all except the excessively rare acute cases with stitutional disturbances recovery can be brought about. The chronic patches tend to persist indefinitely, if untreated, but u treatment disappear in from a few weeks to a few months. The nary type of acute cases rapidly improve upon rest in bed, and they become of the chronic type have the prognosis of that type.

Treatment.—In lichen planus much more can be done by ge measures directed to the improvement of the patients' health tha psoriasis. The patients are usually suffering from nervous overst and require measures directed to overcoming that. Simple life, as out of doors as possible, relief from care, rest, good plain food, t are of service to overcome the systemic depression. Cod-liver oil, qu and strychnin are frequently indicated, and their administration m followed by much benefit.

In all except the acute cases the greatest reliance is commonly p upon the internal administration of arsenic. It is, however, the sensus of opinion of many dermatologists now that arsenic is o less value than mercury. Arsenic is given in the same way as in p sis. Mercury is usually given in the form of corrosive sublimate, p iodide or biniodide, in doses short of producing its physiological

The local treatment of lichen planus is similar to that of psor with the exception that chrysarobin is not so useful and there i usually the necessity for the use of preliminary measures to ge of the scaling. In acute cases the treatment is by the use of soo applications, such as are used in other acute inflammatory disturba lotions like calamine lotion, alone or combined with soothing oint like ointment of oxide of zinc with one-half- to one-per-cent ca acid. In acute cases, in conjunction with these applications, benefit can be gotten from soothing baths, like alkaline, bran, gel or starch baths. In chronic cases the same baths may be useful as vants to other means of treatment.

In the chronic lesions of lichen planus, stimulating application used, taking care to keep them below the point where they produce irritation. The most useful combinations are ointments with ca acid and mercury, of which Unna's ointment is an example:

℞ Acidi carbolici	gr. xx;
Hydrargii bichloridi	gr. ij–v;
Ung. zinci oxidi	℥i.

To be applied several times daily. In spite of the strength of this ointment, it is usually well borne on the chronic patches, but for the sake of caution it is well to begin with a much weaker mixture. The ointment is also made less irritating by increasing the amount of zinc oxide ½ to 1 dram to the ounce.

Tar is also a valuable agent, and is the best application in widely generalized eruptions. It may be used in the form of oil of cade or oil of birch in the strength of ½ to 2 drams to the ounce of zinc-oxide or other ointment. A more agreeable mixture is made by using instead of the pure tar a solution of coal tar, such as Duhring's compound tincture of coal tar. This may be used in strengths of five to fifteen per cent or more in water, applied several times daily, and followed by a nonirritating ointment, or it may be used in the same strengths in ointments. When patches are thickened or warty it is frequently desirable, as a preliminary measure, to soften them. This may be done by prolonged immersion in alkaline baths, and more rapidly by soaking and rubbing them with green soap. In extreme cases it may be desirable to use salicylic acid, ten- to twenty-per-cent strength, in the form of an ointment or plaster, applied to the surface continuously until irritation is produced.

Patches of lichen planus may be improved, and at times made to disappear, by the daily application of high-frequency currents or of a galvanic current of 4 to 10 milliampères.

X-rays, in my experience, have proved very efficient in these cases. They are applied cautiously on alternate days or less frequently, and their effect kept short of producing evident reaction.

LICHEN ANNULARIS[1] (*Galloway*)

SYNONYMS: *Ringed Eruption of the Extremities* (Galloway), *Granuloma annulare* (Crocker).

Under these names very rare cases of ringed eruptions upon the sides and back of the fingers, which are probably a form of lichen planus, have been demonstrated or published by Colcott Fox, Galloway, and Crocker. Hyde also refers to one case in his experience. The affection must not be confused with annular lichen planus, which is a true lichen planus with simply an annular configuration of the patches.

The lesions begin as nodules on the fingers, which spread peripherally into circular or oval patches. They are not reddened, and yet have the appearance of a deep-seated infiltration in the skin. Involution takes place at the center, and there are thus formed round or oval rings, with a center of skin which shows the process in subsidence or which is normal or even slightly atrophic. The lesions attain a size of half an inch to an inch or more in diameter, with a border distinctly elevated, sharply defined, and about an eighth of an inch wide, of ivory-white color, and of doughy or firm consistence. The lesions occur on the sides and back of the fingers, and the exaggeration of the folds of the skin about the knuckles gives the borders a somewhat nodular appearance. The evolution of the lesions is very slow and their course indolent. In Fox's case they had existed two months, in Galloway's three years, and in Dubreuilh's five years. There are no subjective symptoms.

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[1] Galloway, *Brit. Jour. Derm.*, 1899, p. 221 (full consideration with clinical illustration).—Crocker, p. 456.

There is no known cause for the condition. The patients have been both adults and children, and for the most part have been below normal physically. According to Galloway, the lesions closely resemble in their histology lichen planus.

Treatment.—In Galloway's case the lesions disappeared under the application of two- to ten-per-cent salicylic-acid ointment, the administration of iron and cod-liver oil, and improvement in the hygienic surroundings. In Hyde's case, fifty-per-cent aqueous solution of ichthyol was slowly successful after salicylic acid had failed.

Resistant Maculo-Papular Scaly Erythrodermia[1]

Synonyms: *Parakeratosis variegata* (Unna, Santi, Pollitzer), *Dermatitis variegata* (Boeck), *Lichen variegatas* (Crocker), *Dermatitis psoriasiformis nodularis* (Jadassohn), *Lichenoid eruption* (Neisser), *Pityriasis lichenoides chronica* (Juliusberg), *Erythrodermie pityriasique en plaques disséminées* (Brocq).

Under these various names there have been described since 1890 a group of forty-five or fifty cases which present resemblances in turn to generalized forms of psoriasis, lichen planus, and seborrhœic dermatitis, but which have distinctive features that most observers have recognized as sufficiently characteristic to differentiate them from any one of these affections. Clinically they present at times resemblances also to pityriasis rosea, maculo-papular syphilides, the erythematous stage of mycosis fungoides, and pityriasis rubra, but these resemblances are only superficial, and the cases are not regarded as having any essential relationship to any of these diseases.

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[1] Unna, Santi, and Pollitzer, *Monatshefte*, vol. x, 1890; *Abst. Brit. Jour. Derm.*, 1890.—Neisser, *Verhandlungen des LV. Deutschen Derm. Congresses*, in Breslau, 1894.—Jadassohn, *Verhandlungen des LV. Deutschen Derm. Congresses*, 1894; "Festschrift zu *Ehren von Moritz Kaposi*," Wien, 1900.—Juliusberg, *Archiv f. Derm. u. Syph.*, 1897, xli, p. 256; *ibid.*, 1899, l, p. 350.—Brocq, *Journal des Practiciéns*, 1897; *Jour. Cut. Dis.*, 1903 (review of subject).—White, J. C., *Jour. Cut. and Gen.-Urin. Dis.*, 1900.—Fox, C., and MacLeod, *Jour. Cut. and Gen.-Urin. Dis.*, 1901 (good review and bibliography).—Ravogli, *Jour. A. M. A.*, July, 1901.—MacLeod, *Brit. Jour. Derm.*, 1902.—Little, Grahm, *Brit. Jour. Derm.*, 1902.—Meneau, *Jour. Mal. Cut.*, May, 1902.—White, C. J., *Jour. Cut. Dis.*, 1903.—Crocker, *Brit. Jour. Derm.*, 1905, "Xantho-erythrodermia perstans"; *Brit. Jour. Derm.*, April, 1905.—Anthony, *Jour. Cutan. Dis.*, October, 1906.

The cases are probably all of them varieties of one distinct affection. Crocker believes all of the cases are "only variants of one affection, the arrangement of the exanthem being the most variable feature," and that they resemble each other not only clinically, but still more closely microscopically. He, with Jamieson, regards them as closely allied to lichen planus, and has suggested the name lichen variegatus. Colcott Fox and MacLeod believe that the different cases constitute a distinct clinical entity. The name which they have suggested—resistant maculopapular scaly erythrodermias—more accurately describes the affection than do any of the others, and as it predicates nothing as to the character of the affection is adopted here.

Symptomatology.—The affection occurs as a generalized eruption of erythematous, slightly scaly patches. According to Fox, the elementary lesion in all forms of eruption is a small macule or maculopapule, with a fine adherent scale which can be scratched off without bleeding. The larger lesions may arise either from enlargement of these elementary lesions or by their coalescence into patches or figures. In either case, the larger lesions are sharply defined and covered with a sparse layer of thin, delicate, adherent scales. The color of the lesions is pinkish yellow to purplish, and the eruption as a whole has a faint purplish tinge. Upon the lower extremities it is distinctly of darker purplish color. Ordinarily there is little infiltration of the skin, but in the older patches it may be distinct. The individual maculo-papular lesions resemble abortive lichen planus papules. The larger patches in their infiltration and scaliness resemble more nearly a very superficial psoriasis. The eruption is usually generalized and abundant, and may be almost universal, except that usually the scalp, palms, and soles escape, and to a less extent the face.

The configuration of the lesions varies in different cases, and on the basis of these variations Brocq, who regards the disease as a distinct clinical entity which he calls parapsoriasis, divides the cases into three types: (1) Parapsoriasis guttata; (2) parapsoriasis lichenoides; (3) parapsoriasis in patches. This division is at least convenient for purposes of clinical description.

In the first type the eruption occurs as slightly scaly macular or maculo-papular lesions, and resembles an abortive guttate psoriasis.

The second type of cases, because of the peculiar configuration of the lesions, is clinically most striking. The elementary lesions are minute pinhead-sized, flattened papules which coalesce to form peculiar retiform figures inclosing areas of normal skin, and the gene development of this network of lesions gives the surface a peculi characteristic variegated and marbled appearance which is mad

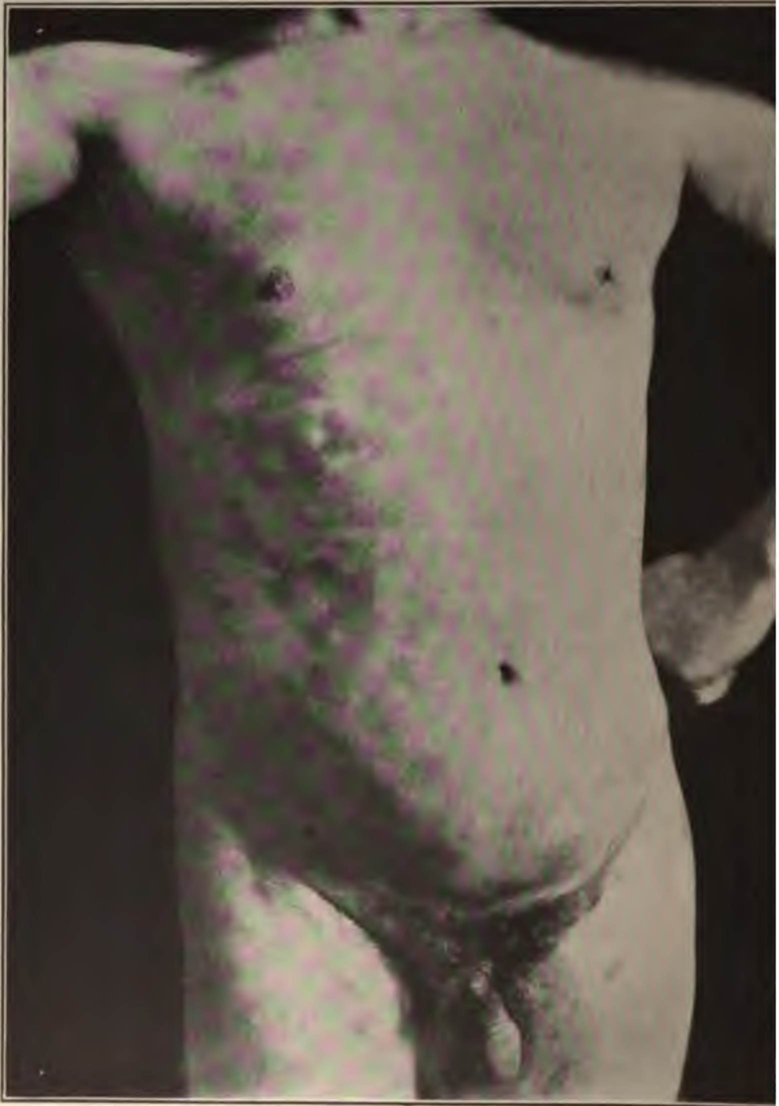

FIG. 112.—PERSISTENT MACULO-PAPULAR SCALY ERYTHRODERMIA. (J. C. White's collection.)

striking by the darker color of the lesions upon the legs. Unde type are included the cases which have been described as *parake* *variegata, dermatitis variegatus, lichen variegatus.* In the second type the lesions resemble more closely those of lichen planus.

In the third type the eruption occurs in sharply defined coin-sized patches of irregular generalized distribution. The lesions show slight branny desquamation; the scales are adherent, but can be scratched off without producing bleeding points, and do not show under scratching the characteristics of psoriasis. In the third type the lesions resemble more nearly those of seborrhœic dermatitis. Under this type Brocq includes the cases described as *érythrodermie pityriasique en plaques disséminées.*

In all of its types the disease is persistent and chronic, lasting unchanged or with exacerbations and remissions for many years (thirty years in Jamieson's case). It frequently improves in summer and becomes more marked in winter. As a rule, there are no subjective symptoms, although occasionally there is slight itching, which is worse at night. The disease is not associated with any disturbance of the general health.

Etiology and Pathology.—Nothing is known of its etiology. It is not contagious or epidemic, and many of the patients are in vigorous health. It is seen in all classes of society. It is most frequent in youth and adult life, but has been seen as early as seven years (Juliusberg). Fox and MacLeod regard it as probably a vasomotor disturbance which is associated with œdema and infiltration of cells in the corium and secondary changes in the epidermis.

Histologically it is a superficial inflammatory process involving the subepithelial layer of the corium with dilatation of the blood vessels, œdema, and cellular infiltration. There is œdema of the epidermis with defective cornification. The epidermis, as a rule, is not thickened, and it may even be thinner than normal. Parakeratosis—in spite of Unna's name for the affection—is not a marked feature of the process.

Diagnosis.—The diagnostic features of the eruption are its persistent character, the thin scales, adherent but not followed by bleeding on removal, the arrangement in patches, or, more characteristic, the reticular arrangement, its general distribution, and its great rebelliousness to treatment. These features are sufficient to distinguish it from psoriasis, generalized lichen planus, and seborrhœic dermatitis, which it most closely resembles.

It may closely resemble a maculo-papular syphilide, in which, however, the history is entirely different, and other evidences of secondary syphilis will be found.

From pityriasis rosea it may readily be differentiated by its persistent character.

In the appearance of the patches and in its persistent course the form occurring in coin-sized patches may be closely analogous to the premycotic stage of mycosis fungoides, but in the latter there is well-marked infiltration of the patches and intense and persistent itching.

Treatment.—The lesions are strangely rebellious to the applications which are usually effective in psoriasis, lichen planus, and seborrhoeic dermatitis. Unna claims successful results from the application of pyrogallic acid, so strong that large doses of hydrochloric acid were necessary to neutralize its poisonous effects. Strong applications of bichloride, either in ointments or lotions, have been of benefit in some cases.

LICHEN SCROFULOSORUM[1]

SYNONYM: *Lichen scrofulosus.*

Lichen scrofulosorum is an affection, occurring in strumous persons, characterized by a more or less widely distributed symmetrical eruption of grouped, indolent, inflammatory follicular papules.

Symptomatology.—The lesions of lichen scrofulosorum are firm, flat, pinhead-sized papules, reddish to dark red in color, and occur around the hair follicles. At first they are bright red, later they become dull red and brownish, and are capped with a minute scale, very rarely by a minute pustule. The papules at the beginning are isolated; later they develop in closely set, coin-sized groups, but they never coalesce. When the eruption becomes fully developed the involved surface is of a dirty yellowish-brown color, and is more or less covered with thin, easily detached scales. The tendency to grouping of the papules is pronounced, and frequently they have a circinate or crescentic arrangement. The eruption is most abundant upon the lower two thirds of the trunk, especially on the sides; less frequently it occurs upon the arms and legs.

The development of the disease is insidious and its course extremely chronic, and it does not usually come under observation until the eruption is well established. The eruption is maintained by the slow evolution and involution of lesions. Older lesions undergo involution and leave minute yellowish stains, which are very slow to disappear, but there is no scarring. The disease as a whole may last for years; it may disappear spontaneously. It occasionally recurs, but after disappearance there may be no recurrence.

It is ordinarily unaccompanied by itching or other subjective symptoms.

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[1] Johnston, *Phila. Monthly Med. Jour.*, February, 1899.—Bronson, *Archives of Derm.*, 1875.—Shepherd, *Canada Med. and Sur. Jour.*, vol. ix, 1880–81.—Currier, *Jour. Cut. and Gen.-Urin. Dis.*, 1892.—Gilchrist, *Johns Hopkins Hospt. Bull.*, 1899.—Crocker, *Brit. Jour. Derm.*, 1899.—Pringle, *Brit. Jour. Derm.*, 1894.—Perry, *ibid.*, 1895.—Fox, Colcott, *ibid.*—Little, *ibid.*, 1900.—Gottheil, *Jour. Cut. and Gen.-Urin. Dis.*, 1886.

Variations.—On the legs and feet there may be hæmorrhage into the papules, producing a condition which has been described as *lichen lividus*.

Sometimes the lesions are large follicular papules. At times these contain sebaceous plugs, and occasionally an acute folliculitis occurs in them, producing pustules like those of acne.

Occasionally many of the papules show horny spines at their tips, producing lesions like those of lichen spinulosus.

At times, in older patients, there is dermatitis around the lesions, especially upon sensitive parts like the scrotum.

Etiology and Pathology.—Lichen scrofulosorum is a disease of childhood and adolescence. It is rare in America, and is most common in Germany and Austria. The disease is clearly associated with tubercular adenitis or with some other form of tuberculosis in a very large proportion of cases (ninety-nine per cent in Austria), and Jadassohn obtained a characteristic tuberculin reaction in fourteen of sixteen cases. Schweninger and Buzzi have seen a similar eruption result from tuberculin injections.

The general and most plausible opinion is that the lesions themselves are not tubercular, but are paratubercular; that is, produced by the presence of tuberculous toxins in the blood.

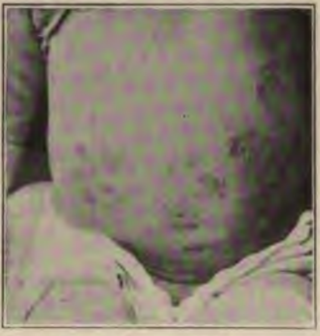

FIG. 113.—LICHEN SCROFULOSORUM; IN PATIENT WITH TUBERCULOSIS OF ANKLE JOINT. (Anthony's photograph.)

Histologically, the papule of lichen scrofulosorum is composed of an infiltration consisting of round cells and large fusiform cells situated around the papillary vessels, and the hair and sebaceous follicles. Most observers have demonstrated the presence of giant cells, but other elements of the typical tuberculous process, such as coagulation necrosis and caseation, have not been observed. Jacobi and Wolff have found tubercle bacilli in the lesions, and together with Pellizarri and Haushalter have obtained positive results from inoculations in guinea pigs. On the other hand, Lafitte, Lefebre, Jadassohn, and Lukasiewicz have failed in similar attempts. Gilchrist has found in one case typical tubercles in the deeper corium beneath the papular lesions.

Diagnosis.—The characteristic features of the disease are the small scaly chronic inflammatory papules around the hair follicles occurring in closely set groups in strumous children. It may be confused with ollicular papular eczemas, follicular papular syphilides, and keratosis ilaris.

In eczemas the lesions are more acutely inflammatory, are very rarely all of papular type, and itching is marked.

The small follicular syphilide is readily differentiated from lichen scrofulosorum by its association with other evidences of secondary syphilis and by its rapid development.

In keratosis pilaris the lesions are horny plugs in the mouths of the hair follicles. They show no tendency to grouping, and are found especially on the extensor surfaces of the arms and thighs.

Prognosis and Treatment.—The disease persists indefinitely if untreated, but readily yields to treatment.

The classical treatment of Hebra, which is rapidly curative, consists of cod-liver oil externally and internally. The oil is administered internally as in other strumous conditions. Externally a daily inunction of the oil is given. For external application the essential factor is the thorough and repeated greasing of the skin, and other oils are about as effective as cod-liver oil.

VI.—FORMS OF GENERAL EXFOLIATIVE DERMATITIS[1]

There are several conditions characterized by dry scaling dermatitis which is generalized and tends to become universal. The various cases have a variable etiology, but tend in the end to produce clinical pictures showing a good deal of similarity, so that there has been no little confusion in the classification of the cases. Among the cases of generalized exfoliative dermatitis, there are several well-defined pathological and clinical entities. These are pityriasis rubra pilaris of Devergie, dermatitis exfoliativa epidemica of Savill, and erythema scarla-

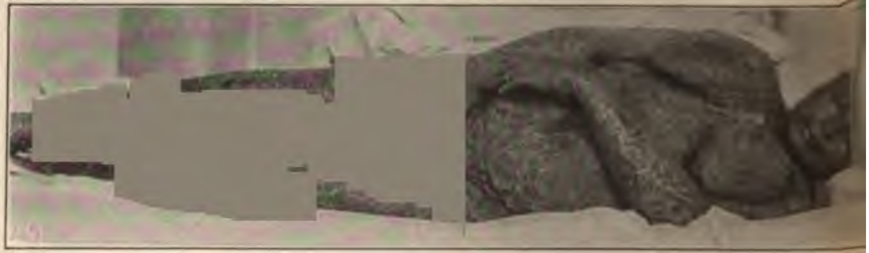

FIG. 114.—DERMATITIS EXFOLIATIVA. (Hartzell's collection.)

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[1] Bowen, *Jour. Cut. and Gen.-Urin. Dis.*, 1902, xx, p. 548.—Brocq, "Erythrodermies exfoliantes généralisées," "La Pratique," t. ii, p. 548.—Bruunsgaard, *Zeitschrft.*, 1901, viii, p. 571.—Luithlen, *Zeitschrft.*, 1902, ix, p. 24.—Török, "Mracek's Handbuch," Bd. i, p. 767.

. Less definite entities are pityriasis rubra of Hebra and s exfoliativa neonatorum of Ritter.

ven after these definite types of general exfoliative dermatitis 1 delimited, there still remain numerous cases which present the picture of generalized dermatitis with scaling, which are s origins and which cannot, perhaps, be included under any of types; these cases are grouped under the disease dermatitis a.

capitulate then: this complicated group of dermatoses char- by general exfoliative dermatitis includes the following:

)ermatitis exfoliativa: a clinical but not a definite pathological

Erythema scarlatiniforme, which probably covers all of the dermatitis exfoliativa except those secondary to other der-

)ermatitis exfoliativa epidemica (Savill's disease).
)ermatitis exfoliativa neonatorum (Ritter's disease).
'ityriasis rubra of Hebra.
'ityriasis rubra pilaris of Devergie.

ema scarlatiniforme has already been described among the ery- ith which by its etiology and pathology it belongs, but it should n mind in considering the other forms of generalized exfoliative s.

f these forms of general exfoliative dermatitis have certain in common. They are all forms of dry dermatitis; about the here may occur linear breaks in the epidermis with slight weep- the fissures, and occasionally the epidermis is broken elsewhere, endency to this is very slight and it ordinarily does not take xcept in pityriasis rubra pilaris the infiltration of the skin ly slight. The color in all forms is an inflammatory red, rom pinkish to darker shades of red. There is in all forms tion. This is for the most part in the form of thin branny flakes, but in the acute types like scarlatiniform erythema may form of large sheets of epidermis. Usually the abundance of a marked feature. In all of the forms there is more or less Rarely it may be severe enough to interfere with sleep and the production of scratch marks. Ordinarily it is moderate stent, and often in the chronic cases its continuous though character greatly disturbs the patient's comfort. In all universal exfoliative dermatitis there is great sensitiveness This applies to all universal eruptions of acute type. It marked in the universal eruptions of chronic inflammatory

Dermatitis Exfoliativa

Synonyms: *Pityriasis rubra, Acute general dermatitis, General exfoliative dermatitis, Dermatitie exfoliatrice, Erythrodermie exfoliante* (Besnier).

Symptomatology.—The cases which are grouped under the term dermatitis exfoliativa are well subdivided by Hyde and Montgomery into three types:

(1) Cases practically identical in origin with scarlatiniform erythema. In their onset they present the picture of scarlatiniform erythema. Either after recurrences or after the first attack the disease may persist as a subacute exfoliative dermatitis which lasts for months or a year or more, but usually ultimately disappears.

(2) The second type is the exfoliative dermatitis of Erasmus Wilson. In this form the disease begins as a dry, red dermatitis which quickly shows abundant exfoliation of thin, usually branny, scales. It may begin around the folds of the joints, the genital region, or on the trunk or extremities. By the formation of new patches it usually becomes quite universal, although it may run its course without involving the entire surface of the body. The color varies from bright to darker shades of red and is somewhat obscured by the abundant scales by which it is screened. The infiltration of the skin is relatively slight. In lax tissues, as the lips and eyelids, there may be some puffiness. As in erythema scarlatiniforme, there may be some free discharge, particularly in fissures in the folds of the joints. The desquamation is abundant, and the scales are rubbed off easily by the friction of the clothing or by the hands. The scales are gray or whitish, usually small and fine, but at times are large and flaky and occasionally sheetlike. There is usually involvement of the hair and nails. There is more or less thinning of the hair, usually on all the hairy parts, but the hair is not entirely lost. The nails may be uplifted by accumulation of horny material under them and thrown off.

The eruption may appear also upon the mucous membranes of the eyes, nose, mouth, and throat, occasionally in the form of gray or red moist macules or patches, covered by a pseudo-membrane. In the acute cases the tongue is usually bright red, undoubtedly from the exfoliation of the epidermis which is washed away by the saliva.

The duration and course of the disease is similar to that of scarlatiniform erythema, although the attacks are usually longer and are more frequently followed by persistent exfoliative dermatitis.

Itching is usually slight or absent, but may be fairly well marked. The disease is frequently ushered in by malaise and more or less acute febrile disturbance, and there may be very considerable prostration. In other cases the general health is not affected.

As will be seen, the resemblances between Wilson's exfoliative dermaitis and scarlatiniform erythema are very close. In Wilson's type the ttacks are somewhat more prolonged and the tendency to persist in-

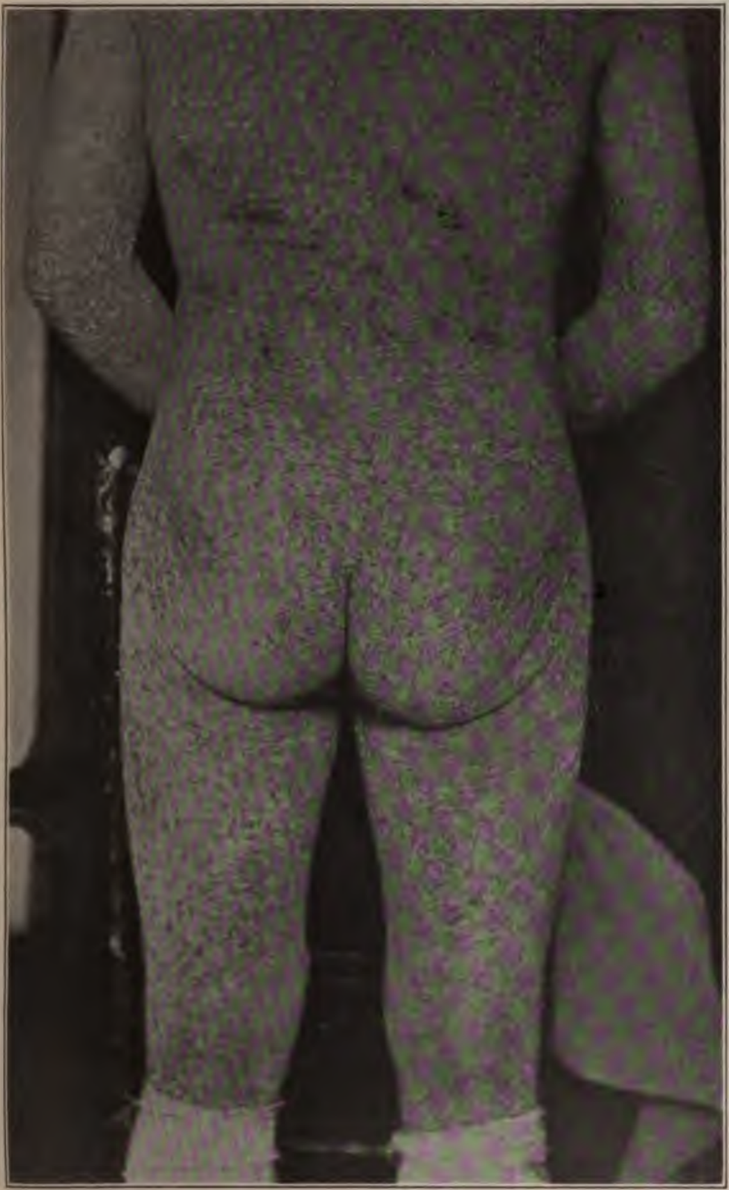

FIG. 115.—EXFOLIATIVE DERMATITIS SUPERVENING UPON PSORIASIS. (Author's collection.)

definitely as an exfoliative dermatitis is more marked, but these are not essential distinctions and, as Bowen puts it, "Its claims to be separated from desquamative scarlatiniform erythema are not very imposing."

(3) The third type includes cases of generalized, usually universal, exfoliative dermatitis which arise secondarily to other dermatoses or from the application of external irritants. Such cases sometimes supervene upon psoriasis, lichen planus, eczema, and upon dermatitis due to external irritants. When such cases become generalized it may be impossible to distinguish them from the two preceding types. When such general forms of exfoliative dermatitis develop from eczema or psoriasis, or lichen planus, it is, as a rule, in my opinion, the result of external irritation, usually by remedial applications. Such a result may occur from ivy-poisoning; I have seen it follow iodoform dermatitis, and in my experience in hospital practice it has not infrequently occurred in psoriasis from the use of chrysarobin. When these universal cases of exfoliative dermatitis result from external applications they usually disappear in the course of a few weeks. Occasionally they persist for a year or more, ultimately recovering. The itching is usually moderate but is sometimes severe. Ordinarily there is no constitutional disturbance.

When any of these types of dermatitis exfoliativa becomes persistent, the picture which is presented is closely similar to that of pityriasis rubra of Hebra. The redness becomes of a deeper hue, and in some cases presents a venous character. There may be at times also marked pigmentation of the skin. The scaling remains abundant, so that the scales can be collected by the handful from the patient's bed. Ultimately some infiltration of the skin develops as a result of the chronic inflammatory process. There is usually no free exudate and perspiration is diminished, so that the skin is dry and harsh. After the cases pass over into this chronic type their distinction from pityriasis rubra of Hebra is difficult, and is, indeed, in my opinion largely a matter of personal opinion as to the significance of names.

Etiology and Pathology.—The etiology of the first and second types is probably the same as that of scarlatiniform erythema, and most of the cases are probably due to some form of toxæmia.

The histological changes are those of an inflammatory process in th corium, with inflammatory œdema of the epidermis and parakeratosis.

Diagnosis.—The first and second of these types of exfoliative de matitis are distinguished from other forms of general exfoliative dermatitis by their more acute onset and course and less persistent characte the third type by its beginning in other inflammatory dermatoses resulting from the application of external irritants.

Pemphigus foliaceus is characterized by the presence of bullæ, abundant discharge of characteristic nauseous odor, and progressive gr prostration; all of these are absent from dermatitis exfoliativa. The affections which it resembles most closely are pityriasis rubra of Hebra and pityriasis rubra pilaris of Devergie.

Prognosis.—The cases usually recover in the course of two or three to several weeks, but the prognosis should be guarded because of the likelihood of recurrence and the possibility of the condition becoming chronic. Some of the cases are said to have ended fatally, but this must be excessively rare. In a case under my observation through two attacks in successive winters the patient, without subsequent attacks, developed chronic interstitial nephritis.

Treatment.—In those cases which are of toxic origin such measures are necessary as are indicated for the toxæmias. In some cases the use of aperients and cathartics is indicated, but as the patients in the toxic cases show more or less prostration, such remedies are to be used with circumspection. Pilocarpine in doses sufficient to produce sweating occasionally gives rapid relief.

For local use soothing applications are beneficial, such as calamine liniment, olive oil alone or with an equal quantity of limewater, zinc ointment, or vaseline. To any of these a minute quantity of carbolic acid, ichthyol, tar, or salicylic acid may be added according to indications. The patients are made comfortable by the use of any nonirritating fatty application. The acute cases should be kept in a warm room, preferably in bed, and care should be taken in applying the local remedies to avoid chilling.

PITYRIASIS RUBRA (HEBRA)[1]

SYNONYMS: *Pityriasis rubra aigu, Dermatitis exfoliativa.*

The name pityriasis rubra (πίτυρον, bran, and *ruber*) is sometimes loosely used as synonymous with dermatitis exfoliativa of Wilson. To avoid confusion it should be limited to the form of persistent general exfoliative dermatitis which was first described by Hebra under the name pityriasis rubra.

Pityriasis rubra of Hebra is a disease characterized by the occurrence of a universal superficial dry desquamative dermatitis, which after becoming once established persists through life without changing in character, although after the disease has continued for several years secondary changes in the skin resulting from chronic inflammation appear.

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[1] Hebra, Hans, Vierteljahr, 1876, Heft 4, S. 508.—Hyde, *Chicago Med. Jour. and Exam.*, 1881.—Petrini & Jadassohn, *Archiv*, 1891, xxiii, p. 961, and 1892, xxiv (complete review and bibliography).—Tschlenow, *Archiv*, 1903, lxiv, p. 21 (bibliography); *Russische Zeitschrift f. derm. u. venerische Krankheiten*, B. 1, Heft 12.—Kopytowski and Wielowieyski, *Archiv*, 1901, lvii, p. 33 (bibliography).—Vietowieyski and Kopytowski, *Jour. des Mal. Cutan. et Syph.*, 1901, Heft 8.—Sellei, *Archiv*, 1901, B. lv, Heft 3.

The disease is very rare. Hebra and Kaposi had together twenty-one cases, and in America cases have been published by Duhring, Fox, and Hyde. One new case has been under my care.

Symptomatology.—Pityriasis rubra is rarely seen until it has become quite or almost universal, but in two cases observed by Kaposi early in their course, it began on the flexor surfaces of the joints as bright red, rather circumscribed patches covered with thin fine scales, withou infiltration and without discharge. The erythema becomes universal i from a few weeks to a year or more. The entire skin is then reddene with little or no infiltration and with abundant free exfoliation of th epidermis. Ordinarily the infiltration early in the course of the disea is so slight as to be imperceptible. Later this characteristic absence o infiltration is lost and more or less infiltration is appreciable. The re ness is that of a superficial dry dermatitis, vivid bright red over mo of the surface, but of darker and bluer tint on the dependent parts. Th redness disappears on pressure, leaving spots which show a yellowis tinge. The exfoliation of horny epidermis is constant in the form o thin small scales or thin flakes, but there is no tendency for the accumu lation of these flakes into adherent masses upon the surface. The erup tion is of the same character upon the head and face as elsewhere. Th palms and soles are paler than the rest of the surface on account of th thick horny epidermis which covers them, and the scales upon these par are thicker and larger and more adherent. Perspiration is less th normal. The temperature of the skin is slightly raised.

The disease is persistent and shows no tendency to spontaneous inv lution. Usually it grows progressively worse, although in the case und my observation there was always marked improvement, with diminuti in the redness of the skin and in the other symptoms during wa weather. Throughout the course of the disease the eruption maintai its uniform type of a diffuse dry dermatitis; it is not an abundant pu tate dermatitis, coalescing into sheets, and there is never, unless it be t result of accidental secondary processes, the formation of vesicles, bul pustules, or crusts.

After the eruption has persisted for two or three years the skin sho the secondary changes resulting from a chronic superficial inflammat process. Sclerotic changes and atrophy of the skin occur. The e dermis becomes thinned, and the skin as a whole more or less harder and infiltrated and less elastic than normal, so that it fits tightly o the surface. The color becomes less vivid and of darker red to purpl and the pigmentation deeper. Late in the course of the disease the co may become markedly livid or even cyanotic. Especially about the l

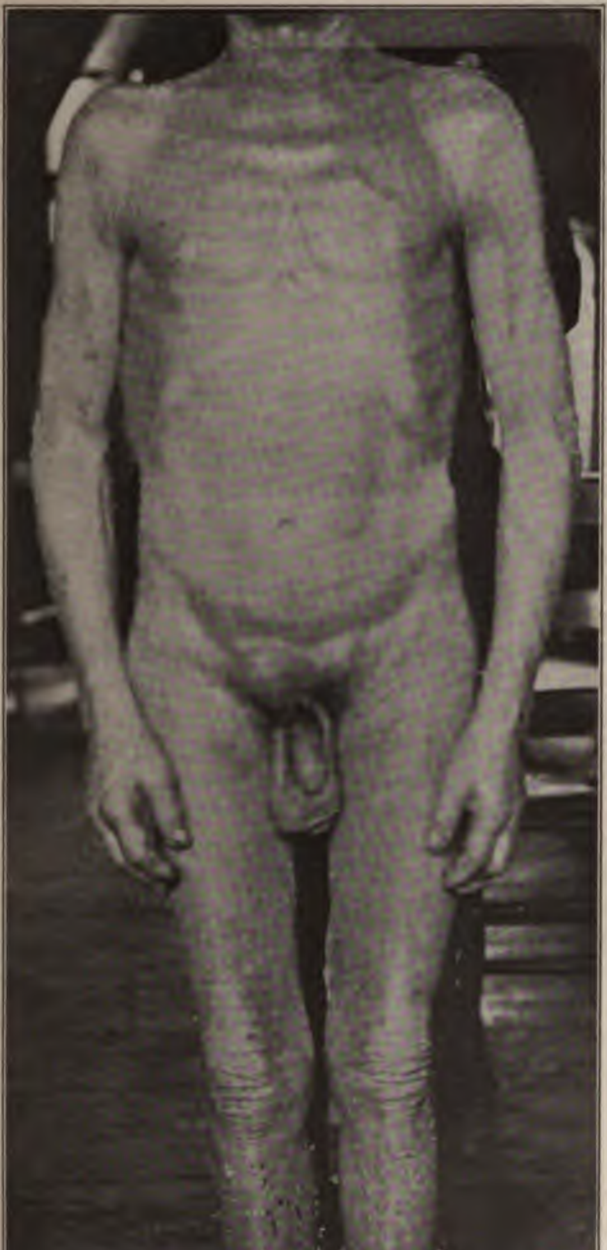

FIG. 116.—PITYRIASIS RUBRA OF HEBRA. (Author's collection.)

there is often very appreciable cutaneous œdema. As a result of these changes the skin is tense and glistening; it can only with difficulty be pinched up into folds; there is some interference with free movement, so that the lips are drawn, the lower lids perhaps ectropic, and the fingers restricted in motion and semiflexed. The exfoliation of the epidermis still persists, but is likely to be less abundant and occur in larger flakes. Upon surfaces where there is much stretching of the epidermis, as over the joints, there are likely to be cracks and fissures, and from thinning of the epidermis on the soles the feet become tender, and walking is made painful. In one case of Kaposi's spontaneous areas of gangrene occurred several times in the course of two years.

Enlargement of the superficial lymphatic nodes is usual, and late in the course of the disease they may show as hazelnut- to pigeon-egg-sized painless masses.

With the complete establishment of the disease the hair and nails become involved. The coarser hairs become fine and short and very much thinned. The lanugo hairs may be entirely lost. There is accumulation of horny epidermis beneath the free border of the nails; the nails become opaque, thin, and brittle, or thickened and rough.

Itching and chilliness are annoying subjective symptoms of the disease throughout its course. During the early period the itching may be slight, but later it is apt to be distressing and interfere with sleep. As in all forms of universal dermatitis, an unusual sensitiveness to cold is a marked symptom.

Early in the disease there are usually no constitutional symptoms. As years go on the patients gradually fail and cachexia appears. In the case observed by me there occurred repeated febrile attacks with œdema of the legs accompanied by pain and extreme tenderness along the inner sides of the thighs. Albuminuria, rheumatism, diarrhœa, and bronchitis are common complications and pneumonia and tuberculosis may complicate the condition. Unless the patients are carried off by some intercurrent acute disease—of which they are the ready prey—they ultimately die from marasmus.

Etiology and Pathology.—Nothing is known of the etiology of the disease. Nearly if not all of the cases are in men, usually between forty and fifty years of age; Kaposi records one dating from childhood.

The anatomy has been studied by Kaposi, Hans Hebra, Elsenberg, Petrini, and Jadassohn. Early in the disease the skin shows the changes of a superficial inflammatory process. In the advanced cases there are the sclerotic changes of a chronic superficial dermatitis. The skin as a whole is atrophied, the interpapillary processes of the rete and the papillæ are distorted or obliterated, the follicles have disappeared, and there is abundant deposit of pigment in the corium.

In the few cases upon which *post mortems* have been made various changes have been found; tuberculosis of various tissues (Hans Hebra, Fleischmann), atheromatous arteries (Kaposi and Petrini), myelitis (Jamieson). Kopytowski and Wielcwieyski have discovered supposedly pathogenic cocci.

Diagnosis.—Kaposi bases the diagnosis upon the exclusion of all forms of secondary exfoliative dermatitis which began in other dermatoses, such as psoriasis, lichen planus, squamous eczema, pemphigus foliaceus, and the chronic erythemata. The characteristic feature of the disease, therefore, upon which it alone can be distinguished from other progressive forms of exfoliative dermatitis, is its development and continuance from beginning to end as one type of eruption.

Prognosis and Treatment.—The disease pursues a progressive course, but it may continue for many years before the patient succumbs. Kaposi thinks he has seen one recent case cured from the internal use of carbolic acid after all local measures had made the disease worse.

The local treatment consists in the use of soothing applications. Baths of various sorts, with alkalies, bran or other demulcents, or tar, may give considerable comfort, after which the body should be freely anointed with a nonirritating salve. It apparently makes little difference what form of fat is used, provided it is nonirritating and is used freely.

The patients should be supported in every way possible, given a liberal diet, and carefully protected from cold. Residence in a warm climate would probably be the most efficient measure in modifying the course of the disease. With the possible exception of the beneficial use of carbolic acid in Kaposi's case no specific internal medication has proved of any value. Arsenic does not benefit the cases.

PITYRIASIS RUBRA PILARIS[1]

SYNONYMS: *Lichen ruber* (Hebra), *Lichen ruber acuminatus* (Kaposi), *Pityriasis pilaris* (Devergie), *Lichen psoriasis* (Hutchinson).

There has been much discussion as to the identity of pityriasis rubra pilaris of Devergie, lichen ruber of Hebra, and lichen ruber acuminatus of Kaposi. According to Kaposi, who was Hebra's disciple and successor, his lichen ruber acuminatus is identical with Hebra's lichen ruber, except that his cases did not show the grave systemic disturbances which characterized Hebra's cases. We may assume, therefore, that the question reduces itself to the identity of pityriasis rubra pilaris and lichen ruber acuminatus of Kaposi.

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[1] Devergie, "Traité pratique des maladies de la peau," 1857, p. 454.—Hebra and Kaposi's "Hautkrankheiten," 1862, vol. ii, p. 315.—Richaud, *Thèse de Paris*, 1877.—Besnier, "La Pratique Dermatologique," vol. iii.—Thibierge, *ibid.*—Kaposi, *Archiv*, vol. xxi, 1889, p. 743, and vol. xxxi, 1895, p. 1.—Neumann, *Archiv*, vol. xxiv, 1892, p. 3.—Neisser, *Verhl. d. Deutschen Derm. Gesell.*, iv; *Trans. Internat. Derm. Cong.*, Paris, 1889.—Fox, G. H., Morrow's "System," vol. iii, p. 324; Discussion, *N. Y. Derm. Soc.*, *Jour. Cut. Dis.*, 1902, p. 572.—Taylor, R. W., *N. Y. Med. Jour.*, Jan. 5, 1889 (valuable paper).—White, J. C., *Jour. Cut. Dis.*, 1894, p. 468.—Zeisler,

The descriptions of these two affections as given respectively by the Austrian and French schools show only minor variations, and that they are identical seems conclusively established "as a result of the last two International Dermatological Congresses, where the same case was claimed as typical lichen ruber acuminatus by Kaposi and other Germans and as typical pityriasis rubra pilaris by different French authorities" (Hyde).

Pityriasis rubra pilaris is a chronic disease of the skin characterized by the formation around the hair follicles of subacute inflammatory papules which are capped by acuminate horny scales. The coalescence of these papules produces infiltrated plaques of dry scaling subacute dermatitis which involves large areas and in many cases the entire surface of the skin.

The disease is rare, but not nearly so rare as typical cases of pityriasis rubra of Hebra.

Symptomatology.—The elementary lesion of pityriasis rubra pilaris is a firm, subacute inflammatory papule, pierced by a hair and capped by an adherent horny spine. The papules are firm and ordinarily of subacute inflammatory red color; occasionally they may be yellowish and show little of the red tinge. When the lesions are discrete the horny tip is deeply inserted into the mouth of the follicle, is firmly adherent, and is white or dirty gray in color. The hair which pierces it may be broken off and found only upon examination with a lens. The disease may appear insidiously, and when first seen show only as a group of such papules, but ordinarily the papules appear in large crops and the disease does not come under observation until there is an abundant widely generalized usually symmetrical eruption. When the eruption occurs in large areas of closely set discrete lesions, they give the surface a peculiar and very characteristic nutmeg-graterlike appearance and feel. The individual lesions have little tendency to peripheral enlargement, but they are so abundant that they tend to coalesce and form diffuse plaques of eruption. Then the skin of the involved areas is very distinctly infiltrated, with exaggeration of the normal lines, and of a bright or dark but not cyanotic red or perhaps of yellowish-red color. In these diffuse patches the scaling loses its spinous character, and there

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Chicago Med. Record, vol. xvi, 1899, p. 533.—Ravogli, *Cincinnati Lancet-Clinic*, vol. xlii, 1899, p. 333.—Heidingsfeld, *ibid.*, June 3, 1899.—West, *Brit. Jour. Derm.*, 1895, p. 273.—Liddell, *ibid.*, p. 279.—Whitfield, *Soc. Trans. Brit. Jour. Derm.*, 1902, p. 470, and 1904, p. 462.—Hall, *Brit. Jour. Derm.*, 1903.—Hügel, *Münch. med. Wochenschr.*, 1900, No. 50.—Rash, *Dermatolog. Centralblatt*, No. 7, 1899.

is an abundant desquamation of fine, branny scales without accumulation into adherent masses. In cases in which the eruption is limited there may be areas of diffuse infiltration and other areas studded with discrete papular lesions.

When limited the eruption is usually abundant and symmetrically distributed, though it may be localized to a few areas. In partial cases areas of predilection are the scalp, face, and hands, the sides of the abdomen, and the extensor surfaces of the extremities. On the face and scalp the eruption occurs in diffuse slightly infiltrated reddish patches. On the face the desquamation is relatively slight and is greasy like that of seborrhœic dermatitis. On the scalp the scales accumulate as a thick, greasy, yellowish mass. On the palms and soles the eruption

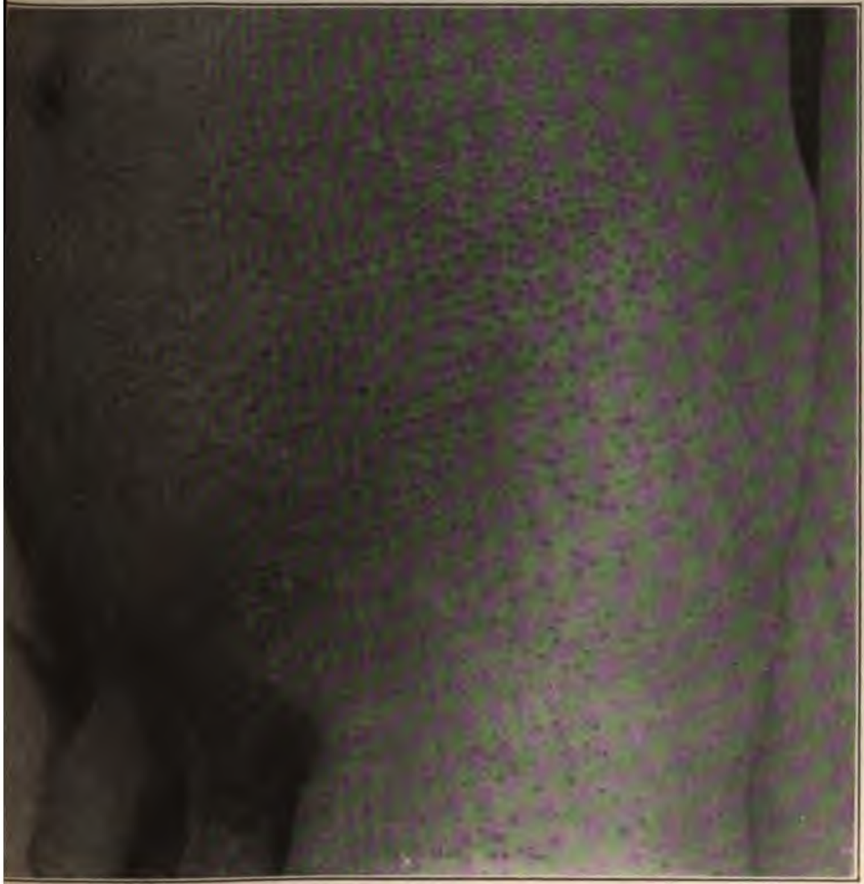

FIG. 117.—PITYRIASIS RUBRA PILARIS; EARLY IN COURSE OF DISEASE, LESIONS DISCRETE. (Author's collection.)

occurs as a diffuse thickening with exaggeration of the normal lines and the formation of thick attached scales. The eruption occurs characteristically around the lanugo hairs on the dorsal surface of the first and

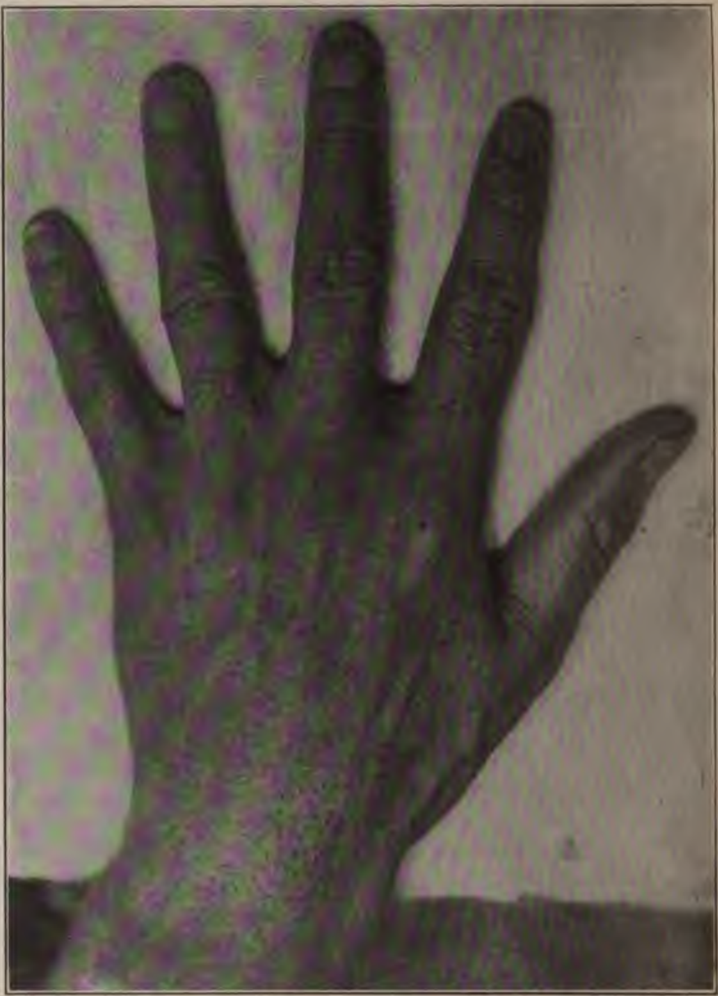

FIG. 118.—PITYRIASIS RUBRA PILARIS. Discrete follicular lesions on back of hand and fingers. (Author's collection.)

second phalanges. Here even in cases otherwise universal the lesio remain discrete and show as peculiar nutmeg-graterlike groups of pa firm, horny papules.

When the eruption becomes universal, as it frequently does, the e tire surface of the skin shows the characteristics of the diffuse patcl already described; the skin is thickened and infiltrated, with exagge tion of the normal lines, of yellowish-red to dark-red color, and with an abundant exfoliation of fine scales. From loss of elasticity fissures are apt to occur about the joints; the face loses its lines of expression; ectropion may occur, and some limitation of the motion of the lips.

The nails usually show nutritional changes and become thin, striated, friable, and stunted. The lanugo hairs are likely to be broken off in the papular lesions, but the coarse hairs on the hairy parts of the body are unaffected. In a universal case under my observation there was complete loss of hair except on the scalp where it was only slightly thinned.

The course of the disease is extremely chronic. It may continue for years without affecting the general health; at least fifteen years in a universal case which I have observed. This patient went through accidentally acquired syphilis without influence upon the dermatosis or upon his health. It may persist unchanged or it may show periods of exacerbation and remission. It occasionally disappears, usually to recur, but in a few cases the relief has been permanent.

The subjective symptoms are very slight. There is very little itching and in the extensive cases some feeling of stiffness in the skin. The sensitiveness to cold observed in most universal inflammatory dermatoses has been much less marked in the universal cases of pityriasis rubra pilaris which I have seen. There are no constant constitutional symptoms. Even in universal cases the patients may remain indefinitely in vigorous health. In some cases there have been failure of nutrition and marasmus, which have terminated in death.

Etiology and Pathology.—The etiology of the disease is not known. It begins, as a rule, during adolescence or early adult life, but has been observed as early as two and a half years of age (Rasch).

The individual papule shows an inflammatory process around the follicle, with thickening of the epidermis and the formation of a horny spine which is deeply inserted into the follicle and projects above it. In the diffuse areas there are inflammatory changes in the corium with thickening of all of the layers of the epidermis and hyperkeratosis. There are no changes in the sebaceous or sweat glands. According to most authorities the primary changes are in the epidermis. According to Kaposi and others the epidermal changes are secondary to inflammation of the corium, and this latter view would seem the more probable one.

Diagnosis.—Pityriasis rubra pilaris may be confused with other dry scaling inflammatory dermatoses, like psoriasis, lichen planus, or pityriasis rubra, and other forms of general exfoliative dermatitis. The pathognomonic features of pityriasis rubra pilaris are the firm, subacute, inflammatory papule with a spiny tip and the characteristic nutmeg-graterlike appearance which is produced by the grouping of the discrete papules. These can always be found on the dorsal surfaces of the second phalanges of the fingers, and, when the disease is limited, around the borders of the diffuse patches.

Neither psoriasis nor lichen planus shows the acuminate inflammatory papules or the nutmeg-graterlike appearance of the lesions, or the almost universal distribution which is found in most cases of pityriasis rubra pilaris. Pityriasis rubra and all other forms of exfoliative dermatitis lack the characteristic acuminate spiny papules and differ further in much less infiltration of the skin.

Prognosis and Treatment.—The disease is rebellious to treatment, but occasionally gets well. It is not established that treatment has any effect upon its course. Arsenic in large doses is highly extolled by Hebra and Kaposi, but as a rule is without effect. Thyroid extract, pilocarpin, and protiodide of mercury have all seemed to be of benefit in some cases. Upon the whole, however, internal treatment is symptomatic, and is directed to improving the general health as far as possible. Cod-liver oil is perhaps the most useful tonic. Heidingsfeld [1] has had good results from arsenic hypodermically, combined with laxatives, intestinal antiseptics, and regulated diet.

The local treatment where there is irritation consists in the use of alkaline, bran, starch, or similar baths given daily or every other day, and followed by the application of bland oils or ointments. To soften the skin and get rid of the horny scales bathing with green soap is useful, to be followed by the use of an ointment. When the process is not irritable slightly stimulating applications may be of service, such as salicylic acid or tar (two to ten per cent) in zinc ointment. In one case of moderate degree under my care considerable persistent improvement has resulted from x-ray exposures carried to the point of producing the first evidences of reaction. In this case there is always great improvement in warm weather, which suggests a possibility that a residence in a warm climate would be beneficial.

DERMATITIS EXFOLIATIVA EPIDEMICA [2]

SYNONYMS: *Dermatitis epidemica, Savill's Disease.*

In the *British Journal of Dermatology* for February, 1892, Savill described a remarkable "epidemic skin disease somewhat resembling eczema and pityriasis rubra which occurred chiefly in the western district of London during the summer and autumn of 1891." The first epidemics were observed in the Paddington and Marylebone and Lambeth infirmaries in the summer and autumn of 1891. Since then other cases have been reported. A very few cases have been reported in this country by Fordyce, Colby, and Winfield.

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[1] *Jour. Cutan. Dis.*, vol. xxiv, No. 8, 1906.

[2] Savill, *Monograph*, London, 1892; *Brit. Jour. Derm.*, 1892, pp. 35 and 69; *Jour. Cut. and Gen.-Urin. Dis.*, 1894.—Colby and Winfield, *Jour. Cut. and Gen.-Urin. Dis.*, 1898, p. 73.—Hutchinson, *Archives of Surgery*, 1891–92, pp. 146 and 221.—Echeverria, *Brit. Jour. Derm.*, 1895, p. 9.—Fordyce, *Jour. Cut. and Gen.-Urin. Dis.*, 1897, p. 141.

Savill describes the disease as "a contagious malady in which the main lesion is a dermatitis, sometimes attended by the formation of vesicles, always resulting in desquamation of the cuticle; usually accompanied by a certain amount of constitutional disturbance, and running a more or less definite course of seven or eight weeks."

There is no doubt that the disease is a distinct clinical entity.

Symptomatology.—The cutaneous manifestations of the disease are those of an exfoliative dermatitis of general and symmetrical distribution. It may begin either as a bright-red follicular papular eruption or as an eruption of erythematous patches. Then by spreading of these initial lesions areas of exfoliative dermatitis are formed. In many cases on the second or third day there is a formation of vesicles on the papules, but the discharge quickly ceases and the process persists as a dry exfoliative dermatitis. In six of Savill's cases the eruption appeared as papules which by peripheral enlargement formed circular red rings with depressed central areas covered with minute vesicles. There is abundant desquamation of scales, under which there is a bright erythema with slight infiltration of the skin. The disease appeared in over fifty per cent of Savill's cases upon the face, scalp, and arms (exposed parts); in seventeen per cent upon the legs. It may appear upon any part. After its first appearance it may remain localized for a time, but usually rapidly spreads. In about half of the cases it remained localized, but in many cases it became universal. The localized cases usually show symmetrical distribution.

The disease runs an acute course of six to eight weeks and may terminate fatally or in recovery. Relapses are common, and occasionally a well-marked second attack occurs. In the cases that recover the exfoliation and the redness lessen and finally disappear, leaving the skin markedly pigmented.

Conjunctivitis is a common accompaniment, as is sore throat. The occipital and cervical glands frequently and the submaxillary occasionally are enlarged without any relation to the eruption upon the head, for this has been observed in cases where the head was unaffected.

There is, as a rule, more or less itching and sometimes it is severe.

The cases usually begin without premonitory symptoms, and where the eruption is not very extensive there may be no constitutional symptoms except slight prostration. There is ordinarily no fever except late in the course of severe and fatal cases. A certain proportion of the cases are ushered in by anorexia, vomiting, diarrhœa, and prostration, and in the extensive and universal cases the constitutional symptoms are usually severe. Albuminuria and diarrhœa are common, as are complications such as pneumonia, gangrene of the feet, and cardiac depression. In the old and in cachectic patients the disease tends to run a severe course, and death has occurred in from five to twenty per cent of cases in different epidemics.

Etiology and Pathology.—The large epidemics were first observed in infirmaries for the pauper sick. The old and the infirm are most susceptible, but occasionally healthy younger individuals and even children have contracted the disease. No defects in sanitation have been discovered to account for the extensive London epidemics. The disease is distinctly contagious. In the first epidemic at Paddington it attacked nineteen per cent of the infirm and sick who were exposed. There was little tendency for it to attack the healthy attendants.

Savill and Russell found in fluid from unruptured vesicles and in scales an organism resembling the staphylococcus albus, but occurring as a diplococcus in rodlike segments, which did not liquefy gelatin and did not produce in experimental inoculations on animals the usual effects of the staphylococcus albus. This is probably the pathogenic organism of the disease.

Savill found the histological changes of ordinary dermatitis. Echeverria described a peculiar degeneration of the nuclei of the prickle-cell layer.

Diagnosis.—The occurrence of an exfoliative dermatitis in epidemics, affecting chiefly the old and infirm and often accompanied by severe constitutional disturbances, clearly differentiates this disease from any similar dermatosis.

Treatment.—Treatment has had little effect upon the course of the disease. Locally the cases should be treated by emollient applications such as are indicated in other forms of acute generalized dermatitis. Crocker reports that in some cases when the disease began in a few patches it has been aborted by painting with tincture of iodine or collodion. The internal treatment is symptomatic and has to do especially with supporting measures to counteract the great depression.

DERMATITIS EXFOLIATIVA NEONATORUM[1]

SYNONYMS: *Ritter's Disease, Dermatitis exfoliativa infantum, Keratolysis neonatorum.*

Ritter gave the name dermatitis exfoliativa neonatorum to a rare disease of the skin characterized by general exfoliative dermatitis, of which he reported 300 cases in the foundling asylum of Prague in ten years.

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[1] Ritter, *Central-Zeitung für Kinderheilkunde*, October 1, 1898; *Archiv für Kinderheilkunde*, 1880.—Elliot, *Amer. Jour. Med. Sci.*, January, 1888, p. 1; Morrow's

The disease is very rare and few cases have been reported in this country.

Symptomatology.—Ordinarily the course is that of a general exfoliative dermatitis, but in a few cases the lesions are moist and suggest a relationship to pemphigus foliaceus or the so-called pemphigus neonatorum. The eruption usually appears in the first or second week of life, occasionally as late as the fifth week. It appears, as a rule, on the lower part of the face, but may appear anywhere. It may begin either as patches of scaling dry dermatitis, or as a diffuse desquamative erythema. In some of the cases the eruption at times shows a vesiculo-bullous stage, but this stage is succeeded as a rule by the usual dry exfoliative dermatitis. The eruption soon becomes universal. It usually involves the mucous membranes of the mouth, nose, and eyes, and fissures occur at the muco-cutaneous junctures as well as, frequently, in the flexures. The disease runs its course in from one to three weeks; the severe cases usually terminate in death within ten days, while the eruption disappears in the course of two to three weeks in nonfatal cases.

The constitutional symptoms may be mild or severe. In the severe cases there are great prostration, with or without diarrhœa, and subnormal temperature; and in the fatal cases (about fifty per cent of the total number) death occurs from marasmus.

Etiology and Pathology.—The etiology of the disease is obscure. Ritter's large number of cases in one institution suggests a contagious character, and he regarded it as of septic origin.

It has been regarded in turn as pemphigus foliaceus (Behrend), as an exaggeration of the physiological exfoliation of the newborn (Kaposi), and as epidermolysis (Caspary). Various organisms have been suggested. Riehl found a mycelial fungus in two cases, Winternitz the staphylococcus aureus and albus in the blood in one case.

Diagnosis.—The characteristic feature of the disease is the occurrence of a universal exfoliating dermatitis in infants within a few weeks of birth, with great prostration and subnormal temperature, and frequently ending in death.

Treatment.—The local treatment consists in the use of emollient oils and fats. The general treatment is directed to supporting the patients in every way possible. Particular attention should be directed to the maintenance of the body heat.

"System," vol. iii, p. 321.—Dorland, *Phila. Polyclinic,* 1896, p. 385.—Patek, *Jour. Cut. Dis.,* 1904, p. 269.

SECTION VI

HÆMORRHAGES

I.—PURPURA[1]

THE term purpura (Greek, πορφύρεος, purple) is applied to cutaneous lesions produced by hæmorrhage into the skin. Purpura is not a disease but a symptom of various disturbances, and when used without qualification signifies simply a hæmorrhagic eruption.

As we have already seen, hæmorrhagic lesions occasionally occur in severe types of various diseases which are characterized typically by inflammatory eruptions of toxic origin. This happens in the different forms of erythema multiforme, in urticaria, in the exanthemata, and in the bullous angioneurotic dermatoses. In addition to these well-defined clinical entities in which hæmorrhage into the skin occasionally occurs, purpuric eruptions are at times an accompaniment of other diseases. In order to comprehend the pathology of these hæmorrhagic diseases of the skin and to classify them properly, they should be compared with the inflammatory angioneurotic disturbances, especially erythema multiforme, to which in their etiology and in the pathological mechanism of their production they are closely related. Indeed, purpura exhibits so many of the characteristics of erythema multiforme that it might well be grouped and considered with that affection.

Classification.—A satisfactory pathological classification cannot be made at the present time. The following provisional classification is taken with modifications from Osler:

(1) Symptomatic purpura:

(*a*) Infectious purpura, as seen occasionally in pyæmia, septicæmia, and malignant endocarditis, and in specific infectious diseases, as in typhus and occasionally in measles, scarlet fever, smallpox, cerebrospinal fever, syphilis, and malaria.

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[1] Osler, *Jour. Cut. Dis.*, 1903; "Practice of Medicine," 6th edition, p. 742.—Bowen, *Jour. Cut. Dis.*, 1892.—Mackenzie, S., *Brit. Jour. Derm.*, 1896.—Weber, *ibid.*, 1900, p. 77.—Engman, *Jour. Cut. Dis.*, 1903.—Burch, *Med. News*, vol. lxxiv, 1899, p. 427.—Howard, *Jour. Exper. Med.*, vol. iv, 1899, No. 2.—Wright, *Lancet*, January 18, 1896; *Brit. Jour. Derm.*, 1896, p. 82; *Brit. Med. Jour.*, December 19, 1891.—Frick, *Kansas City Med. Index*, 1896, p. 159.—MacGowan, *Jour. Cut. Dis.*, February, 1905.

(*b*) Toxic, as from venomous snake bites, which produce rapid extravasations of blood, and from various medicines, especially copaiba, quinine, belladonna, mercury, ergot, and the iodides.

(*c*) Cachectic, such as is seen in cancer, tuberculosis, pseudoleukæmia, leukæmia, Bright's disease, scurvy, and in the debility of old age (purpura senilis).

(*d*) Neurotic, occasionally seen in locomotor ataxia, particularly following lightning pains, also rarely in acute myelitis, in transverse myelitis, and in severe neuralgias. Purpuras also occasionally follow severe emotional disturbances, and rarely occur with hysteria.

(*e*) Mechanical, such as may be seen from traumatisms or from the venous stasis produced by paroxysms of coughing or epileptic attacks.

(2) Arthritic:

(*a*) Purpura simplex.

(*b*) Purpura rheumatica (Schönlein's disease).

(*c*) Purpura, erythema, and urticaria with visceral lesions (Henoch's purpura).

(3) Purpura hæmorrhagica.

Symptomatology.—The cutaneous symptoms of the various forms of purpura do not differ except in degree, and will be sufficiently described in considering purpura simplex and the more definite clinical forms of purpura—viz., purpura rheumatica, Henoch's purpura, and purpura hæmorrhagica.

The essential characteristic of all hæmorrhagic lesions is that they show discolorations, corresponding to the color of blood or blood pigment, which are unchanged by pressure. The explanation of this is, of course, that the pigment and blood corpuscles are free in the tissues, and are not driven out of the tissues by pressure as they are when the capillaries are compressed in an ordinary inflammatory process. Hæmorrhagic lesions may be bright or deep-red color originally, but as the extravasated blood disintegrates the lesions rapidly become dark, and in the course of a few hours or longer change to dark red, then to mahogany brown, and then to dark purple. When the extravasation is least the color of the lesions may be a faint brownish red. From this degree we see, depending upon the amount of hæmorrhage, all gradations of color from brown through mahogany red to dark purple, and in extreme examples purplish-black lesions. Then as the pigment undergoes disintegration and absorption, the lesions slowly fade and go through the familiar involution of a bruise; from purple or brownish red to dark greenish yellow, to brownish yellow, and then to the normal color of the skin. In nearly all instances the lesions eventually disappear in the course of two or three weeks. On the legs they are frequently followed by pigmentation which is permanent or persists for years.

Depending upon their size and outline, the lesions of purpura are designated as petechiæ, vibices, ecchymoses, and ecchymomata. The distinctions between them are all unessential, and all of them may be

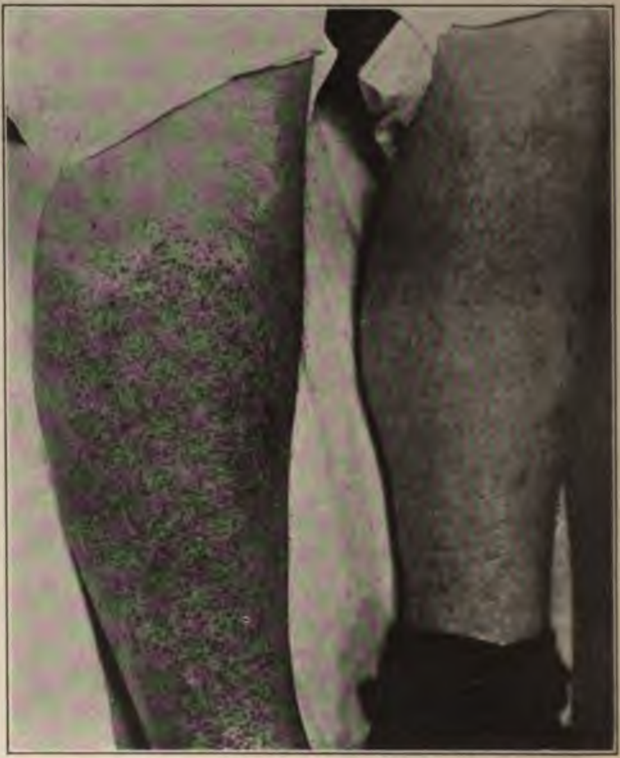

FIG. 119.—FOLLICULAR PURPURA. (Heidingsfeld's collection.)

found in a single case. Petechiæ are hæmorrhagic macules, usually pinhead to tackhead size, occasionally larger, and of roundish or irregular shape. Occasionally the extravasation is sufficient to cause elevation of the skin so that the petechiæ are maculo-papules. Vibices are hæmorrhagic streaks. Ecchymoses are hæmorrhagic patches, they are usually of irregular outline, and may be of any size from coin-sized patches to vast extravasations covering an entire part. Duhring and Stelwagon have recorded cases of purpura simplex in which circinate lesions wit clear centers occurred. Ecchymomata are lesions in which the extravasation is so abundant as to form tumorlike swellings; they are cutaneo hæmatomata. Rarely, in association with the ordinary purpuric lesion hæmorrhagic bullæ are formed. In purpuric lesions the borders may sharply defined, but they usually fade out into the healthy skin, and large ecchymoses one may see all variations of intensity of these lesions from the almost black central extravasation through purple, greenish-purple, brownish-yellow to normal color.

Lesions may occur upon the mucous membranes in all forms of purpura. These are common in severe forms, and may be accompanied by hæmorrhage not only from the mucous orifices but in internal organs.

The distribution of purpura usually shows distinctly the effects of gravity; occasionally also the effects of friction and pressure. The sites of predilection of hæmorrhagic eruptions are the lower extremities, especially the inner sides and lower half of the thighs, and the legs; next most frequently they are found on the forearms. They may appear anywhere, but are rare upon the face. I have, however, seen an extreme purpura in acute leukæmia located chiefly on the face, and sharply confined to the surface above the diaphragm.

The lesions of purpura are usually abundant and show symmetrical distribution. After their first appearance they show ordinarily no tendency to peripheral extension, and unless the lesions become confluent upon their first development they show no peculiar configurations arising from coalescence of adjacent lesions. The lesions, however, are of themselves altogether irregular in shape.

All purpuric lesions as a rule appear suddenly and fade out very slowly. A lesion will reach its full development in from a few hours to a day, and then gradually go through the stages of disintegration. The duration of a single lesion is from two to three weeks. Purpuric eruptions as a whole do not usually pursue any definite course. The lesions appear irregularly, and in this way the eruption may be extended indefinitely.

Hæmorrhagic lesions themselves are, as a rule, free from subjective symptoms. They do not itch; they may be tender and occasionally are quite so. When accompanied, as is not infrequent, by urticarial lesions, these itch as usual. Purpura may occur without any constitutional symptoms. However, in the mildest cases of purpura simplex there are, as a rule, lassitude and malaise, and the severer forms of purpura are symptoms of grave constitutional conditions.

Purpura Simplex.—In Osler's classification purpura simplex is grouped with the arthritic purpuras, but although frequently associated with rheumatic symptoms it is also frequently symptomatic of other toxic disturbances, and is better, therefore, regarded as a symptomatic purpura.

Purpura simplex is a term used to designate the mild cases in which the lesions appear as petechiæ and small ecchymoses. The eruption may last only for a couple of weeks. It shows some tendency to persist, and in rare cases it is continued indefinitely by the constant appearance of new crops of lesions. Cases persisting for two or three or even five years (Stelwagon) have been observed. In rare instances the purpuric lesions are accompanied by lesions of erythema multiforme or of urticaria (*purpura urticans*), but these are much less frequent than in the severer forms of purpura. The cases of purpura simplex accompanied by rheumatic symptoms are in all probability mild types of purpura rheumatica.

Purpura Rheumatica.

SYNONYMS: *Peliosis rheumatica, Schönlein's Disease.*

This affection is characterized by multiple arthritis and purpura along with lesions of urticaria and erythema multiforme. There may also be nodular lesions indistinguishable from those of erythema nodosum and, exceptionally, vesicles or bullæ. Along with these other lesions there may be extensive areas of angioneurotic œdema (giant urticaria) which may or may not be ecchymotic; these cases have been described as *febrile purpuric œdema.* In short, we have here a disease showing all forms of lesions transitional between the inflammatory angioneurotic disturbances like erythema multiforme and purpura.

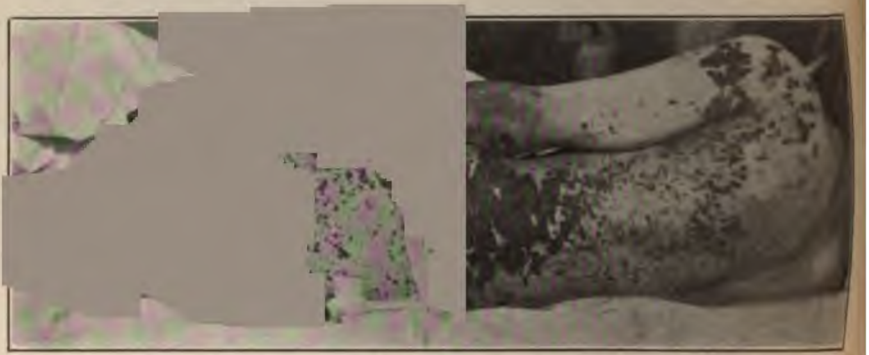

FIG. 120.—PURPURA RHEUMATICA. Note several hæmorrhagic bullæ on back. Lesions in mouth, with a large slough of tongue. (Author's collection.)

The disease usually begins with a temperature of 101° to 103° F., with articular pains, and not infrequently with sore throat. The arthritis is usually slight, the constitutional symptoms severe. There may be endocarditis, pericarditis, and other symptoms of rheumatism, the picture on the whole in most cases being that of acute articular rheumatism plus purpura. The disease may run a fatal course, but according to Osler the cases usually do well and a fatal result is extremely rare. The throat symptoms may give trouble, and Osler has seen sloughing of the uvula in two cases. Some cases show a tendency to annual recurrence, thus further resembling erythema multiforme.

The combination of multiple arthritis, purpura, and urticaria furnishes a syndrome which is characteristic of purpura rheumatica.

Henoch's Purpura.—This is purpura combined with erythema, urticaria, and visceral lesions. It occurs chiefly in children, and according to Osler is characterized by: (1) Relapses or recurrences often extending over two years; (2) purpura, urticaria, angioneurotic œdema, and erythema multiforme, the skin lesions varying greatly in different attacks; (3) gastrointestinal crises; (4) moderate joint symptoms; (5) hæmorrhage from the mucous membranes; (6) enlargement of the spleen; (7) nephritis, which is its most serious feature and often the cause of death. It is thus an affection almost surely of infectious origin, closely related to Schönlein's disease, if indeed any definite line of demarcation exists between them.

Purpura Hæmorrhagica.

Synonyms: *Morbus maculosus Werlhoffii, Land-scurvy.*

This term is applied to severe cases of purpura with hæmorrhage **from the** mucous membranes. The eruption appears after a few days **of malaise** in the form of ecchymoses upon the trunk and extremities **and at** points exposed to pressure and friction, and the eruption **rapidly becomes** abundant. The lesions appear upon the mucous **membranes,** and are accompanied by bleeding from the mucous surfaces. There is usually slight fever but considerable prostration, and in some cases the general symptoms simulate typhoid fever. Profound anæmia may rapidly develop, and the patients may die from loss of blood or from cerebral hæmorrhage. According to Osler, in favorable cases the disease terminates in from ten days to two weeks. According to other observers its course is slow, and the disease at times disappears only after several months. In rare severe cases, usually in children, purpura hæmorrhagica pursues a very malignant course which may prove fatal within twenty-four hours—*purpura fulminans.* Cutaneous hæmorrhages develop, but death may occur before any bleeding appears.

Purpura hæmorrhagica has been observed in epidemics (Hyde and Montgomery), and is in all probability an infectious disease.

Purpura hæmorrhagica has to be diagnosticated from scurvy, from which it differs in the absence of swelling and ulceration of the gums, and in the absence of the history, usually obtainable in scurvy, of inability to obtain proper food.

Purpura Senilis.—This term is applied to the purpuric lesions which occur about the legs of the debilitated and old. They appear about the ankles and knees and recur from time to time. The condition is purely local, and is usually associated with sluggish peripheral circulation or with varicose veins. It is a very common condition.

Purpura Pulicosa.—This term is applied to the punctiform hæmorrhagic lesions which are produced by the bites of fleas, bedbugs, and other animal parasites.

Etiology and Pathology.—The causes of purpura have been hinted at in the classification given above. In nearly all forms of the condition there is strong presumption that the disease is caused by some sort of toxæmia. It may be from ingestion of drugs, from snake bites, from infection, or from metabolic disturbances. Osler suggests that most of the purpuras are produced by "some poison—an alkaloid, possibly the result of faulty chylopoietic metabolism, which in varying doses in different constitutions excites in one urticaria, in a second peliosis rheumatica, and in a third a fatal form of purpura."

The hæmorrhage takes place chiefly into the corium, but may extend to varying depths into the subcutaneous tissue. Immediately after extravasation the red corpuscles may be found in masses in the interstices of the corium. Later in their stead are seen pigment granules which are gradually absorbed. The dilated capillaries may be blocked with red corpuscles, and there may also be found in the tissues the usual changes of inflammatory œdema. Much study has been given to the changes in the vessel walls in purpura, but without uniformity in the findings. Various conditions have been noted—endarteritis, lardaceous degeneration of the walls, the formation of thrombi and emboli, and plugging of the vessels with microörganisms. The red corpuscles find their exit from the vessels in some cases by diapedesis, in others through rents in the vessel walls. There are no uniform changes in the blood. In some forms of purpura the coagulation time is retarded to ten or fifteen minutes (Osler) in comparison with three to five minutes as the normal.

Diagnosis.—The characteristics of hæmorrhagic lesions in the skin have already been referred to, and the diagnosis of purpura requires simply the recognition of these. The diagnosis of purpura, however, is but the recognition of a single symptom, and is simply an intimation of the various conditions which underlie it. The ultimate diagnosis must depend upon the recognition of the characteristics of the various diseases which produce it.

Treatment.—The internal treatment of purpura is symptomatic. In general tonics, good food, and fresh air are indicated. In all except the mildest cases the patients should be kept in bed. In purpura simplex of children or where there are slight articular symptoms, Osler recommends arsenic pushed to the physiological limit. In checking the bleeding, aromatic sulphuric acid, ergot, turpentine, tannic or gallic acid may be useful. Oil of turpentine in 10- or 15-minim doses three or four times a day is recommended by Osler and Crocker. Wright recommends calcium chloride in 20-grain doses four times a day for not longer an three or four days. Iron and quinin are frequently useful, the latr especially in malarial purpura. Some cases are apparently of syphitic origin and yield rapidly to mercury and the iodides. The salicytes are disappointing in the arthritic forms (Osler). MacGowan,[1] rom very favorable effects in severe symptomatic purpuras, as in urtiaria and erythema multiforme, highly recommends the use of adrenalin olution. He advises it in 10-drop doses of 1:1,000 solution every two ours, either hypodermically or by the mouth. This may be continued or forty-eight hours and in smaller doses for many days. His results are such as to highly recommend the treatment. In a cachectic purpura in a three-and-a-half-pound baby, he gave with excellent results half a drop of 1:1,000 solution every two hours for a week. To check the bleeding from the mouth and nose inhalations of carbon dioxide or irrigations with two-per-cent solution of gelatine or with a 1:1,000 or 1:5,000 solution of adrealin are beneficial.

The lesions of the skin require no local treatment. The legs should be kept in a horizontal position, and friction and pressure avoided as much as possible. In severe cases Besnier recommends the use of light compression with roller bandages.

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[1] *Jour. Cut. Dis.*, February, 1905.

SECTION VII

INFECTIOUS DISEASES OF THE SKIN

The infectious diseases of the skin constitute a very large and important group. A considerable number of them with different clinical manifestations are due to the common pyogenic organisms; others are due to specific organisms which cause characteristic and peculiar clinical pictures; still others of undoubtedly infectious character are due to organisms which as yet are undiscovered. Most of these diseases of infectious origin have been included here in this class. It seems more convenient, however, to consider a few of the diseases of bacterial origin elsewhere. For example, it seems conducive to clearness to consider the exanthemata with the erythemas, because of the similarity of their cutaneous lesions to other toxic erythemas, and to consider various infectious diseases of special structures, like acne (folliculitis of the sebaceous follicles) and sycosis (folliculitis of the hair follicles), and the infectious diseases of the nails, with the diseases of these structures.

DISEASES PRODUCED BY PUS ORGANISMS

As may readily be understood, pus organisms are likely to contaminate all diseases of the skin in which the horny epidermis is broken, and this secondary infection of the various ulcerative diseases of the skin is a condition which constantly demands our consideration. This is, perhaps, the most important rôle of pus organisms in diseases of the skin, but in addition they are the primary causes of a considerable number of dermatoses, among which are

Impetigo,
Ecthyma,
Pemphigus neonatorum,
Pemphigus contagiosus,
Erysipeloid,
Erysipelas,
Granuloma pyogenicum,
Boils,
Carbuncles.

There are a good many other dermatoses which are probably, but less frankly, the result of infection with the common pus cocci.

In speaking of the common pus organisms reference, of course, is made to the staphylococcus aureus, albus, and citreus, and to the streptococcus pyogenes, and these are the usual pathogenic agents in pustular dermatoses. In certain suppurative dermatoses, as in acne, chancroid, and erysipelas, special but closely similar cocci have been described, but in almost none of these instances has it been definitely established that the special organism is not one of the common cocci. It is well established, however, that there are certain pathogenic organisms which occasionally produce suppuration in the skin without contamination with the common pus cocci. Among these are the ringworm fungi, blastomyces, actinomyces, and the tubercle bacillus, but the suppurative conditions which they produce in the skin are quite distinct from those produced by the common pus organisms, and have characteristic peculiarities.

It is not always possible to distinguish clinically the conditions produced by streptococci and those produced by staphylococci, and it is still less easy to distinguish clinically those produced by different forms of streptococci. Thus impetigo may be produced by both staphylococci and streptococci; and, as Jordan has shown, erysipelas may be produced not only by the streptococcus pyogenes or the streptococcus of Fehleisen, but also by staphylococci. Indeed, it is apparently true that the different clinical pictures produced by the pyogenic organisms in the skin depend chiefly upon the site of infection. Thus in impetigo we have a superficial infection of the skin producing a dermatitis; in boils the infection takes place through the follicles and produces a deep-seated suppurative inflammatory process in the connective tissue around the follicles; in erysipelas the process involves the deeper connective tissue of the skin and spreads diffusely along the lymphatics. All of the different conditions, however, have the common features, that they are suppurative inflammatory processes, are due to the same pathogenic agents, and are auto- and hetero-inoculable.

I.—IMPETIGO[1]

SYNONYMS: *Impetigo contagiosa, Impetigo simplex, Impetigo vulgaris, Porrigo contagiosa, Impetigo streptogenes, Impetigo staphylogenes, Impetigo circinata, Impetigo figurata.*

History.—In the early days of dermatology the formation of pus in the skin was regarded as a special disease to which the term impetigo

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[1] Fox, Tilbury, *Brit. Med. Jour.*, 1864; *Jour. Cut. Med.*, 1869; "Treatise on Skin Diseases," Second American Edition.—Duhring, "Cutaneous Medicine."—Bockhart, *Monatshefte*, 1887.—Sabouraud, *Annales*, 1900.—Wickham, *Brit. Jour. Derm.*, 1892.—Elliot, *Trans. Amer. Derm. Assn.*, 1899; *Jour. Cut. and Gen.-Urin. Dis.*, 1894.—Gilchrist, *Trans. Amer. Derm. Assn.*, 1899.—White, C. J., *Trans. Mass. Med. Soc.*, 1899.—Corlett, *Cleveland Jour. of Med.*, 1898.—Allen, *Trans. Amer. Derm. Assn.*, 1896.—Anthony, *Jour. Cut. and Gen.-Urin. Dis.*, 1898.—Engman, *ibid.*, 1901.—Grindon, *ibid.*, 1901.—Schamburg, *ibid.* 1896.—Klotz, *ibid.*, 1896.

(from *impetere*, to attack) was applied. From this loose usage Tilbury Fox rescued the term when he applied it to the well-defined symptom complex which he called impetigo contagiosa. Since Fox's description of impetigo contagiosa there has been considerable diversity of opinion as to whether this was a specific disease or a condition which presented no essential differences from superficial pustular lesions in the skin resulting from accidental inoculation with any of the common pus organisms. Unna would divide impetigo into impetigo vulgaris (impetigo contagiosa), impetigo circinata, impetigo staphylogenes, impetigo streptogenes; Sabouraud divides it into impetigo due to streptococci (impetigo contagiosa), and impetigo due to staphylococci (impetigo of Bockhart). All this is, there seems little doubt, academic refinement which cannot be justified by any essential differences in the different forms of impetigo or by any clinical differences which serve to distinguish them. As Bockhart has shown, the condition which we describe as impetigo can be produced both by the common streptococci and by staphylococci and, as for special varieties of impetigo, such as impetigo figurata and impetigo circinata, they simply refer to occasional peculiarities of configuration which are not essential characteristics. The different forms of impetigo, then, may be regarded as part of one clinical condition, and the term may be used quite as well without qualifications to describe the pustular and vesicular lesions which are produced in the skin by infection with the common pus organisms.

Symptomatology.—The lesions of impetigo may be at first vesicles which later become pustular, or they may be pustules from the start. The vesicles or pustules arise from an inflammatory base, are rather large (up to the size of a split pea or larger), are usually rather flaccid, but may be tense, and soon after their development they begin to flatten down with the formation of a crust at the center, while they spread somewhat at the periphery. In the course of a few days the lesions become covered with yellowish or yellowish-brown crusts, or rupture and leave an abraded red surface from which there is an exudation of serum and pus which dries into crusts upon the surface. These lesions heal after a few days, the crusts are thrown off, and a temporary red stain is left.

The vesicles and pustules of impetigo usually increase in size after their appearance. In this way a single pustule may develop into a good-sized bulla, perhaps tense and oval but usually soft and flaccid. This flattens down into a crust around which there may be a pustular border which tends to creep and still further enlarge the lesion. In this way individual oval lesions the size of a finger nail or larger may be formed. The large bullæ of impetigo, however, usually result from the confluence of adjacent lesions and are of irregular outline.

etigo may consist of no more than two or three discrete pus-
h go through the evolution outlined above. Ordinarily the
sists of one or more patches at different sites of inoculation.
es are made up of
esions which coalesce
irregular areas of
rmatitis of the size
or larger, around
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brownish pus crust,
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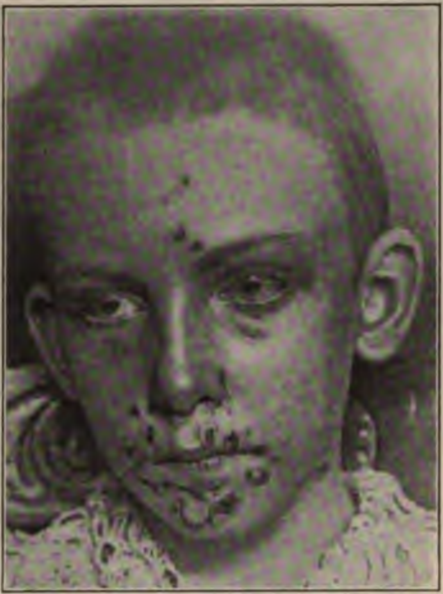

FIG. 121.—IMPETIGO, WITH A TENDENCY TO FORMATION OF ANNULAR LESIONS. (Author's collection.)

go may occur wher-
is an abrasion. It
t frequently on those
abrasions are com-
d exposure to infection greatest. It is therefore seen most
on the face, especially around the nostrils and mouth. A
ght impetigo is the pustular infection that frequently occurs
lt of infection of fever blisters. Because of its production
tching which head lice induce, it is very common upon the
eruption may, as already said, consist of only a few pustules,
spread almost completely over the head and face and occur
ands and feet and, to a certain extent, over the body gen-

rse of the disease is indefinite. A single patch of eruption will
f itself in seven to ten days if no new inoculation occurs, but
will continue as long as inoculations take place. It frequently
or weeks and even months, and becomes very extensive where
r is not recognized and proper treatment is not instituted.
hing associated with the lesions is inconsiderable; that is its
ction from patches of eczema which become secondarily in-
a rule there is no constitutional disturbance, but where the

disease is very extensive there is moderate fever from absorption of septic toxins.

Variations.—Some of the lesions of impetigo may not go on to suppuration, so that we may have cases in which, together with pustular lesions, there is a greater or less abundance of inflammatory papular lesions. In certain cases, apparently chiefly as result of lowered resistance of the patients, the lesions are mostly bullous, *impetigo bullosa.*

In some cases the lesions tend to spread widely at the periphery, and with this extension new peripheral pustules form. Thus lesions of crescentic or circular outline will be formed, and where many such lesions occur upon a surface they coalesce and produce fantastic polycyclic figures (*impetigo circinata seu gyrata*). Crocker speaks of gyrate impetigo as rare, but in my experience the tendency to circinate arrangement is not uncommon and certainly should not confuse one. Circinate impetigo is readily distinguished from the other serpiginous pustular

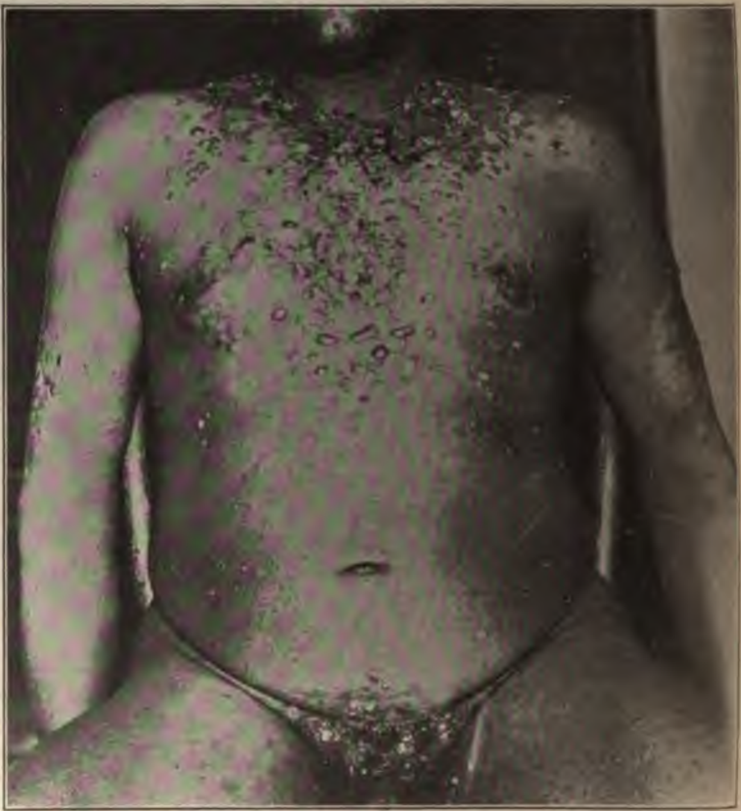

FIG. 122.—IMPETIGO BULLOSA OF UNUSUAL EXTENT, ALSO INVOLVED GENITAL AREA. (Author's collection.)

eruption which occurs commonly, ulcerating serpiginous syphilide, by the fact that there is no destruction of the corium and no scarring.

The so-called *impetigo of Bockhart*, where the lesions occur around hairs, often produces primarily tense, globular, yellow pustules varying in size from minute pustules up to those as large as a pea. This form of impetigo occurs most frequently on hairy parts, especially upon the bearded part of the face. A typical picture of it is the impetigo of the face, which develops from infection around the hairs as the result of shaving. According to Bockhart this form of impetigo is always due to infection with staphylococci, but even accepting that this is a universal fact, Bockhart's impetigo presents no essential clinical difference and no distinction of practical importance from simple impetigo.

Etiology and Pathology.—About seventy-five per cent of cases of impetigo occur in young children. It is most frequent in the poor and uncleanly, but it is not confined to any class of society. All that is required for its production is an abraded surface and the presence of pus organisms. It starts frequently in abrasions like fissures and cold sores about the mouth and nose, and it is a common sequel of parasitic diseases which cause scratching. It is frequently caused in men by the abrasions which are produced in shaving.

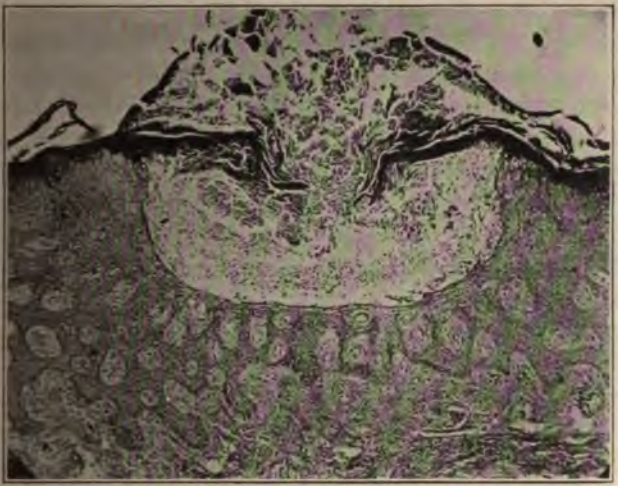

FIG. 123.—BOCKHART'S IMPETIGO. (C. J. White's collection.)

Impetigo is auto- and hetero-inoculable. It is contagious and frequently occurs in epidemics. Epidemics of impetigo are frequently excited through the exchange of clothing and the common use of towels (*football impetigo, bathhouse impetigo*). It not uncommonly occurs with vaccination, as it may with any other focus of suppuration.

The studies of Crocker, Bockhart, Gilchrist, C. J. White, Corlett, and others have established the fact that impetigo may be produced by either the common streptococci or staphylococci. It is most frequently produced by the staphylococcus aureus. Crocker first described cocci in unruptured vesicles, and later showed that some of the cases were due to the staphylococcus aureus. We are chiefly indebted to Bockhart's investigations for the establishment of the fact that the disease has no specific organism, but in all of its forms is a manifestation of infection of the superficial layers of the skin by any of the common pus organisms.

The lesions are the result of a very superficial inflammatory process involving chiefly the epidermis and the papillary layer. The pustules are formed between the horny layer and the rete. There is no ulceration of the connective tissue and no scarring follows it.

Diagnosis.—The most important practical matter in a case of impetigo is the recognition of its character; that being done the treatment is a simple matter. If one remembers that the lesions are such as may be produced by an infection of any superficial abrasion—as, for example, a cold sore—it would seem that the recognition of the condition would be easy, but the fact remains that its character frequently escapes detection. The characteristic features of the disease are that it begins with inoculation of an abraded point, its lesions are those of pustular dermatitis, and the process is so superficial that scarring does not follow the lesions.

It must be distinguished from pustular eczema, sycosis, ulcerating syphilides, chicken pox, and pemphigus.

In a pustular eczema we have a secondary infection of an already existing dermatitis, so that practically we have an impetigo superimposed upon an eczema. With the removal of the infection the eczema is left. If it is an eczema of local origin and the cause no longer acts, the eczema may go on rapidly to cure. If it is an eczema of internal origin, in addition to the patches of pustular eczema, other eczematous lesions which are not pustular will in all probability be found upon other parts. But the distinction between a pustular eczema and an impetigo is one largely of names as far as the lesions are concerned.

In sycosis there is a pustular folliculitis deeply involving the hair follicles, in some of which the hairs are loose and the follicles distended with pus. There is swelling corresponding to the depth of the inflammatory process. In impetigo the pustular dermatitis is on the surface of the skin and does not involve the follicles deeply, and there is no swelling.

In ulcerating syphilides with crusting there is destruction of the connective tissue and scar formation, both of which are absent in impetigo

:hicken pox the lesions are smaller, do not occur in patches, are
l, and have a regular distribution; the disease pursues a regular
and is accompanied by some constitutional symptoms.

pemphigus the bullæ occur suddenly and spontaneously with
no inflammatory areolæ, are entirely independent of abrasions
culations, their contents are at first likely to be sterile, and the
is not contagious. Most of the epidemics of bullous eruptions are
f bullous impetigo.

distinction between ecthyma and impetigo is considered under
ı.

gnosis.—Nothing is easier to treat than impetigo if its char-
recognized, and if the causes of the abrasions which become in-
l can readily be removed. The only cases which are troublesome
ey are recognized are those which are produced by scratching,
g from some irremovable or undiscoverable cause.

atment.—The treatment of impetigo consists in cleaning the
of pus and dirt and the subsequent use of antiseptic applications.
there is much crusting and suppuration these may be removed
ous dressings of boric acid and vaseline. Usually the surface can
ned more readily by thorough washing, until all crusts are re-
with boric-acid solution or weak bichloride solution, or similar
ic solution. After this there should be applied two or three times
an antiseptic ointment. The ointment of ammoniated mercury,
0 grains to the ounce of vaseline or cold cream, is usually em-
and leaves nothing to be desired. If the disease is on the scalp,
uld be looked for carefully and destroyed, and then the lesions
as elsewhere. In extensive cases over the body antiseptic baths
be given until the lesions are cleaned, and then ointments applied.

II.—ECTHYMA

impetigo we have infection of the skin which results in superficial
r lesions without destruction of connective tissue. In the same
infections of the skin a more intense inflammatory reaction may
luced, as a result of lowered resistance of the tissues, with the
ion of ulcers. To these ulcerative pustular lesions of the skin
ig from infection the term ecthyma is applied. It is not a dis-
isease. A superficial ulcer on the shin, such as a careless boy
t from an infected abrasion, may be roughly taken as an illustra-
an ecthymous ulcer.

esion of ecthyma begins as an infected abrasion or as a flaccid
. If the lesion begins as an infected abrasion, ulceration takes

place in the abraded surface and a dirty pus crust forms over a superficial ulcer which has an inflammatory border. If it begins as a pustule the contents dry into a crust, under which there forms a superficial ulcer which is surrounded by an inflammatory areola. The ulcers of ecthyma vary from the size of a finger nail up to a coin, and their depth corresponds to a certain degree with their area. The lesions may be single but are usually multiple, and are frequently associated with typical lesions of impetigo. Their common location is upon the legs, their occurrence being favored by the poorer circulation in the skin of those parts. After the legs they occur most frequently upon the buttocks, but in individuals of poor resistance they may occur upon any part of the body. The course of the condition is uncertain. The lesions may recur indefinitely in persons of lowered resistance who fail to have proper treatment or in the dirty.

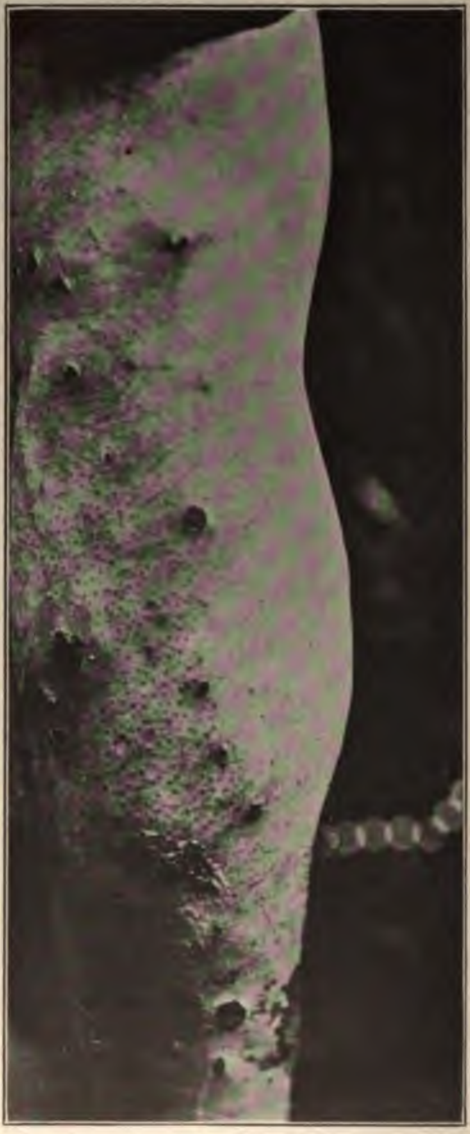

FIG. 124.—ECTHYMA.
(Grover W. Wende's collection.)

Etiology and Pathology.—Ecthyma is most frequent in individuals of lowered resistance. It is seen as a rule among the poor and the dirty, and is a common vagabond's disease. It is due to the same organisms that cause impetigo, and the mechanism of its production is the same as that of impetigo, with the added factors of lowered resistance of the tissues either from cachexia or poor circulation.

Diagnosis.—Clinically it is distinguished from simple impetigo by he fact that it is a more deep-seated process, and is accompanied by lceration, but it is frequently associated with typical impetigo on other arts.

The ulcers of ecthyma on the legs have to be distinguished from yphilitic ulceration of the legs. They are readily distinguished from hose of syphilis by the absence of all of the characteristic features of syphilitic ulcers (*q. v.*).

Treatment.—The lesions of ecthyma may readily be cured with ordinary antiseptic dressings. Wet antiseptic dressings are the best application. If these are not convenient to use, the lesions should be cleaned by washing with bichloride solution or other antiseptic solution and then dressed with an antiseptic ointment, like ammoniated mercury, 20 to 40 grains to the ounce of vaseline.

VELD SORE

SYNONYM: *Natal sore* (?).

This is an infected ulcerating lesion which has come into notice since its frequent occurrence in British soldiers during the Boer War. The descriptions given of it are those of an ecthyma exaggerated by the heat and dirt of semitropical conditions. Crocker suggests that it is of this haracter, and there is nothing in its descriptions or bacteriology which hrows any doubt upon this supposition. It occurs as ecthymous ulcers, specially on the hands and forearms, feet and legs, and is readily cured ith weak antiseptic applications.

III.—PEMPHIGUS NEONATORUM[1]

SYNONYMS: *Pemphigus acutus neonatorum, Pemphigus contagiosus, Pemphigus epidemicus.*

Pemphigus neonatorum is a term which has been applied to a form f bullous impetigo occurring in infants and associated with constituonal symptoms of sepsis.

Symptomatology.—The eruption occurs as more or less abundant rops of bullæ which have slight, if any, areolæ, and may or may not beome purulent; in rare cases they become hæmorrhagic (Crocker). The isease usually appears within the first two weeks of life, and the lesions evelop without any regular sequence. Constitutional symptoms are the rule, and grave septic infection, perhaps followed by fatal issue, is not uncommon. The occurrence of the disease is always associated with circumstances predisposing to septic infection. It appears under bad hygienic surroundings and before the days of asepsis occurred as epidemics in lying-in institutions; in some instances outbreaks have been traced to a special midwife. The disease is usually seen with other septic conditions, such as puerperal fever, mammary abscess, and impetigo in other members of the family.

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[1] Richter, *Dermatolog. Zeitschr.*, vol. viii, 1901 (complete review of the subject).—Maguire, *Brit. Jour. Derm.*, 1903.—Adamson, *ibid.*, 1903.—Crary, *Jour. Cutan. Dis.*, January, 1906 (bibliography).

Pathology.—Its identity with impetigo has been established not only by numerous bacteriological studies, but by the production of impetigo in adults from pemphigus neonatorum in cachectic babies. I most of the investigations of the lesions the staphylococcus aureus h been found; in some the streptococcus pyogenes.

Diagnosis.—There are essential resemblances between pemphig neonatorum, dermatitis gangrænosa infantum, and, perhaps, Ritter's di ease, and these diseases should be studied with reference to each oth The diagnosis of pemphigus neonatorum, as of other bullous forms impetigo, can readily be made from the spontaneous bullous eruptio like dermatitis herpetiformis and pemphigus, by the fact that the co mon pus organisms can readily be cultivated from the bullæ of impetig while in pemphigus and dermatitis herpetiformis the bullæ are at fi sterile. Pemphigus neonatorum has to be diagnosticated especially fro bullous congenital syphilis. The distinction is easy because of the a sence of other evidences of congenital syphilis which are always prese in infants developing bullous syphilides within two weeks of birth.

Treatment.—These bullous forms of impetigo contagiosa are treat in the same manner as other forms of impetigo. The patients must removed from the possibility of further infection, placed under as go hygienic conditions as possible, and the lesions treated by the use of ant septic baths, ointments, or powders. The constitutional treatment symptomatic.

IV.—INFECTIONS FROM VARIOUS PUS-FORMING BACTERIA

Pustular infections of the skin resembling those due to common pu organisms have resulted from infection with various special bacteria.

Diphtheria of the Skin.—Various cases of pustular infection of th skin of the type of impetigo or of ecthyma have been found in whic the organism proved to be the Klebs-Loeffler bacillus. The diagnos from simple impetigo or ecthyma is only made by bacteriological exa ination. In these cases diphtheria antitoxin is indicated.

Pyocyaneus Infections.—A number of cases of pustular infections the skin from bacillus pyocyaneus have been described by Adamso Pernet, and others.

Bacillus Coli Infection.—Auché has described multiple abscesses due to bacillus coli in the skin of an infant.

Vacciniform Ecthyma of Infants.—This condition, which has been especially studied by the French, consists of an eruption of large ecthymatous pustules which soon show central depression from the formation of a crust, and thus form lesions closely resembling vaccine pustules. The lesions occur by predilection about the genitals, thighs, and buttocks; less frequently on the hands and feet. They are apparently produced by infection with streptococci.

V.—FURUNCLE

SYNONYMS: *Boil, Furunculus.*

A furuncle (Latin, *furunculus,* a petty thief) is an acute inflammatory process around a follicle of the skin produced by infection through the follicle and resulting usually in necrosis and suppuration of the central mass.

Symptomatology.—A boil begins as a painful, hard induration, situated deep in the corium, over which the skin quickly becomes an inflammatory red. This rapidly forms an acuminate inflammatory swelling with a central point which is usually either the mouth of a sebaceous or a hair follicle. The swelling increases for from two to six days. At the end of three to six days there develops around the follicular opening a pustule which ruptures with slight discharge. Sometimes the inflammatory process is not intense enough to produce necrosis, and the boil subsides more or less rapidly (blind boils).

There is always a possibility of autoinoculation around boils, so that satellite pustules, each pierced by a hair, are common (these, by the way, are typical lesions of Bockhart's impetigo). In addition to these impetigo pustules, not infrequently other boils occur around the original lesion, being produced by pus carried externally from the parent boil to the neighboring follicles, or by subcutaneous extension of the process. While single boils are common, it is not infrequent for boils to be multiple, and in some cases they are very numerous. This is particularly true in depressed individuals in whom precautions are not taken to prevent autoinoculation. With the occurrence of a boil, as in other infectious lesions, neighboring glands are likely to swell and may suppurate.

Boils occur most frequently, for obvious reasons, upon hairy parts which are exposed to friction. The commonest locations are first the nape of the neck, then the nates and the axillæ, but they may occur anywhere.

As is well known, the pain of boils is usually severe. It is produced by the tension in the tissues caused by the intense inflammatory infiltration, and varies, therefore, with the density of the tissues. A boil in lax tissues, like the cheek, may be almost painless; in tissues like those of the hands and feet, the perineum, the nose, and the external auditory canal, where there is no opportunity for swelling, the pain is severest. It is almost instantly relieved when the tension is overcome by free incision, and subsides more gradually upon the natural opening of the lesion. The constitutional disturbance corresponds in degree with the extent of suppuration and of absorption of septic toxins. With a single boil which is opened promptly there may be the most evanescent or no febrile reaction. Usually there is slight increase in temperature with malaise, and where boils are numerous there may be a fairly sharp septic fever. In the gravest cases there may be pronounced general sepsis.

Hydradenitis Destruens Suppurativa.—Infection in boils usually occurs in the pilo-sebaceous follicles. It may, however, occur in the sweat glands, and to this form of furuncle Verneuil has given the name hydradenitis destruens suppurativa. Such boils present no essential or important differences from those in the hair follicles, and the name should not be allowed to cause confusion. The typical boil of this sort is the common, rather small boil of the size of a hazelnut which occurs in the axillæ. The lesions are usually multiple and are very frequently accompanied by surrounding impetigo pustules. These boils are commonly associated with excessive sweating. They are most frequent in the axillæ, but also occur in the genitocrural region, on the face and neck and back, and they might, of course, occur wherever there are sweat glands, although their existence is not recognized on the palms and soles.

Etiology and Pathology.—The essential factor in the production of boils is the presence of pus organisms. We therefore frequently see them associated with impetigo, suppurating eczema, and other suppurative diseases of the skin. Inoculation may take place from other individuals, and hence association with persons having suppurating lesions of any sort, or lack of care in keeping the skin clean, may explain their occurrence. They very commonly follow local injuries. Boils may occur in individuals of normal resistance, but they are most frequent in those who are not in vigorous health, and their repeated occurrence is usually an indication of some defect in the individual's health. They are apt to develop in individuals who are reduced by overstrain of any sort, and are common in those suffering from chronic alcoholism, anæmia, chronic nephritis, and especially diabetes mellitus. They occur not uncommonly after acute illnesses, particularly the specific fevers. They are more frequent in men than in women.

As the researches of Garré, Bockhart, and others have shown, boils are practically always due to the staphylococcus aureus, albus, or citreus. It is conceivable, however, that they may be produced by other pus organisms. They have been produced artificially by rubbing pure cultures of the staphylococcus aureus and albus into uninjured skin.

Treatment.—In patients subject to boils the health should be investigated carefully, and every effort made to build up their resistance. Improvement of the conditions of living should be provided for, if possible; there should be ample provision made for fresh air, sunlight, and suitable exercise. There is constant demand in these cases for tonics, among which iron is most frequently needed, and after that strychnine, quinine, and arsenic. Where there is a predisposition to the occurrence of boils in the gouty, the use of colchicum is highly recommended. In chronic furunculosis it is especially important to be on the guard for diabetes, nephritis, and anæmia. Specific internal treatment directed to the overcoming of furunculosis offers little prospect of help. The drinking of fresh brewer's yeast, from one to two ounces daily in several doses, has been recommended by Brocq and Crocker and others, and is sometimes beneficial. Calcium sulphide, which, following the suggestion of Ringer, has been used in all sorts of suppurative conditions, has, in my experience, proved useless. Improvement of the individual's resistance by Wright's method of treatment by "vaccinations" with dead cultures of the specific organisms is a rational suggestion and worthy of trial. I have seen it apparently beneficial in acne, and it would be more strongly indicated in furunculosis.

At the beginning of a boil there is some hope of aborting it by local treatment. This can sometimes be done by pulling out the central hair, passing a fine probe dipped in carbolic acid into the follicle as deeply as possible without causing pain, and then touching the apex of the boil with liquid carbolic acid. Another method which is frequently successful is the application in the early stage of the boil of Unna's carbolic acid and mercury plaster. Another method is injecting the base in the early stage of the boil with a drop of carbolic acid or with five drops of 1:30 solution of carbolic acid. Occasionally beginning small boils may be made to disappear by painting with plain collodion, but if this causes pain from pressure it does no good and should be removed. Gallois and Courçon advise for the abortive treatment of boils that the lesions be painted once or twice daily with a solution of metallic iodine four parts in acetone ten parts, acetone being used as a vehicle because of the great solubility of iodine in it. Bulkley recommends for its soothing qualities the following ointment:

℞ Carbolic acid	gr. v–x;
Fl. ext. of ergot	ʒi–ij;
Powdered starch, Zinc oxide, āā	ʒij;
Rose ointment	℥i.

To be applied freely on a thick dressing of absorbent cotton.

For the relief of pain at the beginning of a boil the application of compresses as hot as can be borne is useful. There is some difference of opinion as to the advisability of opening boils early. There is nothing, however, which relieves pain like free incision, and, if it is evident that a boil is going to suppurate, nothing would seem to be gained by waiting for it to point. A boil should be incised freely enough to provide for perfect drainage and relief of tension, and the incision should, in order to avoid scarring, be made in the lines of cleavage of the skin. The best way to reduce the pain of incision is to freeze the part with ethyl chloride. After incision the cavity should be syringed out with carbolic-acid solution, 1 : 30, and as much of the necrotic tissue removed as possible. In emptying it, however, care should be taken not to squeeze it or otherwise injure the tissues. Finally, it is a good plan to swab out the cavity with pure carbolic acid. After incision the best application is a wet dressing of weak bichloride or carbolic-acid solution or of boric-acid solution.

During the existence of a boil attention should be directed to preventing inoculation of other points and the production of new boils. To this end the common poultices should never be used. Their place is perfectly supplanted by hot sterile or antiseptic compresses. It is well to wash the area around the boil with a strong antiseptic solution, such as 1 : 1,000 bichloride in alcohol, and occasionally it may be a good plan to protect the surrounding skin with an antiseptic ointment, like salicylic acid 20 grains, or ammoniated mercury 40 grains to the ounce of simple ointment.

In patients who suffer from recurrence of boils much can often be done by systematic attempts to sterilize the skin as far as possible, particularly that of the axillæ and other parts of the body where boils are most prone to occur. For this purpose it is a good plan to have the patient take baths of bichloride 1 : 5,000 or 1 : 10,000, and to use antiseptic soaps, such as one-half-per-cent bichloride soap, or the other mercurial soaps which are obtainable in the market. At the same time the axillæ, the nape of the neck, and other points where boils frequently develop can be washed daily with 1 : 2,000 or 1 : 5,000 solution of bichloride and alcohol. This is a plan of treatment which I have found very useful.

VI.—CARBUNCLE

SYNONYM: *Carbunculus.*

A carbuncle (Latin, *carbo,* a live coal) is an acute inflammatory lesion analogous in its pathogenesis to a boil, but involving the tissues more diffusely, and ending usually in a more or less extensive slough which finds its exit through multiple openings in the skin. A carbuncle may be regarded as a boil which, either from a greater extent of infection, or from infection of greater virulence, or from infection of tissues of lowered resistance, results in a much more extensively destructive inflammatory process than occurs in boils. Carbuncles are not rare, but are very much less frequent than furuncles.

Symptomatology.—A carbuncle begins as a flattened red induration of the skin and subcutaneous tissues, accompanied by tension and dull throbbing pain. The affected area is much more extensive than that in boils, but is usually sharply circumscribed. The process increases in intensity rapidly, the swelling becomes greater, the skin becomes dark red or purplish, and the pain and constitutional symptoms become more severe. If its course is unaltered by treatment, after five to six days pustules appear upon the surface and rupture, with a bloody, purulent discharge. There is then sloughing of the skin at multiple points, and beneath these openings there is seen a peculiar characteristic ashen-gray slough. The slough may later be discharged through the several openings, or the openings in the skin may enlarge until a single ragged ulcer is formed with a necrotic floor. With the breaking down of the skin the slough slowly separates, and after its separation healing takes place by granulation. The slough usually is limited by the subcutaneous fascia, but may go deeper.

Carbuncles are usually single. The amount of tissue involved in the inflammatory process is seldom smaller than the palm, and it may be much larger. The sites of predilection for carbuncles are, in order of frequency, the nape of the neck, the shoulders, the face, and the abdominal walls. The most dangerous location for carbuncles is the scalp and face, where there is the greatest danger from sepsis and thrombosis and embolism. Carbuncle occurring upon the upper lip and cheek (malignant facial carbuncle) is especially dangerous, on account of the free anastomosis of the blood vessels of the part with those of the meninges, and requires prompt radical treatment.

A carbuncle usually reaches its full development in from eight to ten days. Its entire duration varies according to the size of the lesion and the method of treatment employed. The slough may separate and healing take place within two weeks, but the process may be drawn out over five or six weeks in severe cases.

The pain and the constitutional symptoms in carbuncle are severe. The lesion begins with a feeling of great tension and throbbing pain, which rapidly increases until it becomes agonizing. It was described by Gross as pain like that of molten lead upon the surface, and it has also been compared to such pain as would be produced by an enormous claw imbedded in and dragging on the skin. There is always considerable systemic disturbance as result of sepsis. Severe cases are characterized by great prostration, weak pulse, rigors, and high temperature, and not infrequently in such cases death from exhaustion or from septicæmia occurs.

Variations.—As we have blind boils, so, in rare instances, we have carbuncles in which the tissues become boggy and are infiltrated by a septic inflammatory process, but breaking down does not occur. In one case in the writer's knowledge such a carbuncle without sloughing spread over the nape of the neck, was accompanied by severe constitutional symptoms, and only yielded upon complete excision.

In very severe cases the skin over the central area of the carbuncle may become gangrenous *en masse*. It becomes bluish black, a hæmorrhagic blister forms, and the skin breaks down into a pultaceous mass.

Etiology and Pathology.—Carbuncle occurs most frequently in later adult life and oftener in men than in women. It is a disease of individuals of lowered resistance, the causes which predispose to it being similar to those which predispose to furunculosis. Diabetes in particular is a predisposing cause of it. Carbuncles, like furuncles, are due to infection with staphylococci through the follicles of the skin. As the result of more virulent infection or of poorer resistance the inflammatory reaction is more intense and extensive, and spreads widely through the subcutaneous tissues.

Diagnosis.—Carbuncles are distinguished clinically from furuncles by the larger area involved, the flattened, brawny induration, the multiple openings, and the graver constitutional disturbance. When occurring about the face they may be confused with anthrax. In anthrax there is more rapid progress with the quick development of gangrene, and the bacillus of anthrax can be demonstrated in the pus.

Prognosis.—Carbuncle is a grave disease. The danger increases with the extent and virulence of the process, and an index of it is furnished by the severity of the constitutional symptoms. Where the process spreads widely, and in the aged and those reduced by disease, the outcome is uncertain. This is especially so in diabetics and in carbuncle occurring on the face or scalp.

Treatment.—The internal treatment of carbuncle is directed to counteracting sepsis and supporting the patient in every way possible. The diet should be liquid but nutritious, and the bowels should be kept open with mercurial or saline cathartics. In nearly all cases the prostration is best met by the use of alcoholic stimulants with quinine and iron and strychnine. One of the best ways of administering alcohol in such cases is in eggnogs. If pain is severe it should be relieved as far as possible by the free use of morphine.

During the early stage, before evidence of suppuration appears, hot fomentations frequently changed should be applied. These may be made with 1:40 carbolic-acid solution, or 1:1,000 bichloride solution, or with lead and opium wash. As in furuncles, poultices should not be used. In the early stages the spread may sometimes be stopped by the subcutaneous injection at numerous points around the carbuncle of a few drops of a 1:30 carbolic-acid solution (Crocker). As soon as any evidence of suppuration appears the lesions should be freely incised and free drainage provided for. Cavities should be syringed out with carbolic-acid solution, 1:40, or swabbed out with pure carbolic acid. In severe cases the slough may be curetted out under an anæsthetic, and the cavities then syringed out with carbolic-acid solution or swabbed out with pure carbolic acid and lightly packed. In many cases the surgical removal of the entire mass is the best treatment. It reduces the danger, saves time, and greatly diminishes the amount of suffering.

A method almost as radical as that of curetting or excision consists in the vigorous use of caustics. A method recommended by Crocker is to insert carbolic-acid crystals into all of the openings. A more vigorous method which is highly recommended by Phillips of Wisconsin, from a personal experience and from an experience in a number of cases, consists in the insertion into each opening of a piece of caustic potash. This is left for about twenty-five minutes, until cauterization is thorough. After this the cavities should be washed out with weak solution of acetic acid to neutralize the caustic potash, and wet antiseptic dressings of carbolic-acid solution (1:40) applied. The relief of pain and the improvement within twenty-four hours after this treatment, provided it is carried out thoroughly, are said to be remarkable.

VII.—ERYSIPELAS[1]

SYNONYM: *St. Anthony's Fire.*

Erysipelas is an acute specific inflammation of the skin and subcutaneous tissues, and sometimes of the mucous membranes, which is characterized by the formation of red, swollen areas that tend to spread at the periphery and have little or no natural tendency to the formation of pus, to ulceration, or to gangrene, and end in spontaneous resolution without scarring. It is an infection of the lymphatic spaces of the corium and subcutaneous tissue.

Erysipelas is one of the common diseases affecting the skin.

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[1] Miles, "Article on Erysipelas." "Encyclopædia Medica."—Duplay et Reclus, *Traitè de Chirurgie*, vol. i, 1897 (full bibliography).—Kaposi, *Wien. med. Wochenschr.*, 1887; *Archiv*, vol. xx, 1888.—Fehleisen, "Die Ætiologie des Erysipelas," Berlin, 1883.—Pawlowsky, *Berlin. klin. Wochenschr.*, 1888, p. 255.—Pfahler, *Phila. Med. Jour.*, January 13, 1900.—Marmorek, *Compt. Rend. Soc. de Biol.*, 1895.—Cotton, *Boston Med. and Sur. Jour.*, 1899 (full bibliography of treatment with Marmorek's serum).—Baum, *Medicine*, 1899.

Symptomatology.—Erysipelas begins, after a period of a few hours to one or two days of malaise and moderate febrile disturbance, with the appearance of one or more red spots at the site of infection, which spread and swell and form a large, red, inflammatory patch that is tense, smooth, glistening, and hot. Its temperature is distinctly raised above that of the unaffected skin. The outline of the patch is usually irregular, but its borders are sharp. From it red lines, indicating the inflamed lymphatics, may radiate to the contiguous glands, which become enlarged and tender, and later the swollen, deep lymphatics may feel like large cords. As the process becomes more intense the color changes to a dark, angry red, the swelling becomes greater, the surface more glistening, and vesicles and bullæ filled with clear yellow serum may develop upon it. The swelling varies not only with the intensity of the process but with the character of the tissues. In lax tissues it is much greater, so that when occurring upon the face or the scrotum the swelling may be enormous. After reaching its full development a patch may show no disposition to enlarge, but commonly there is a tendency for the disease to spread at the border while it subsides behind, thus presenting the picture of a migrating cellulitis. In its spread erysipelas does not follow the direction of the lymph stream, but rather the lines of cleavage of the skin, and it is frequently checked by the deep furrows. The face and head are by far the most frequent site of erysipelas, but no part is exempt. On the face it usually begins in abrasions about the nose; thence it spreads over the face and scalp. It may spread into the mouth or into the nose or pass over the scalp, but it usually stops at the chin and nape of the neck, and very seldom involves the front of the neck. It often stops at the border of the hair or beard. The area involved in the disease may not be more than the size of a hand. Frequently it will extend over a large part of the face and head; it may involve an entire limb, or in extreme cases cover a large part of the body.

Occasionally erysipelas develops in surgical wounds. In such cases the process often subsides without suppuration taking place in the wound and healing goes on uneventfully; occasionally pus forms and the edges of the wound separate.

Erysipelas sometimes occurs in the newborn, usually from infection in the navel, and in such cases is apt to be fatal.

As shown by Fehleisen's therapeutic inoculations the incubation period of erysipelas varies from fifteen to sixty hours. The inflammatory process attains its maximum within a few days to a week after its appearance, remains at the maximum for one or two days, and then begins to subside. The entire course varies from a few days to several weeks, the prolonged cases resulting from continual migration of the

ase or from the development of new areas of disease. Relapses are
quent. When the process begins to subside the swelling gradually be-
nes less, the redness decreases, finally changes from red to brownish
, then to yellowish, and then disappears. With the subsidence of
process there is greater or less desquamation, usually in large scales
flakes. Where there have been bullæ these dry up, epidermis forms
der them, and the dead epidermis is thrown off in flakes.

The subjective symptoms are burning and more or less pain and
nderness in the involved area. This tenderness is greatest, as long as
e process is spreading, in a zone about half an inch wide just outside
e red border, this zone of tenderness representing the actively spreading
rder of the process. Its existence is an important clinical point as
dicating the persistent activity and the direction of spread of the
sease.

The constitutional symptoms are those of an acute febrile disturbance
om toxæmia. These symptoms begin, from half a day to two days
efore the appearance of the lesions, with a feeling of malaise, pain in
e head, back, and limbs, loss of appetite, and perhaps nausea and
omiting. There is chilliness, but, as a rule, no rigor. With the begin-
ing of these symptoms there is a slight rise in temperature which
aches by the time of the development of the lesions 103°, 104°, or
05° F. The temperature continues high with moderate evening
acerbations throughout the course of the disease. It falls quickly
hen the process is checked, usually between the fifth and tenth days.
severe cases the return to normal is gradual. After the temperature
s once subsided, its subsequent sharp rise usually means a new in-
lvement of the disease. A constant high temperature without remis-
n is a grave symptom, indicating often complications, such as sup-
rative cellulitis, gangrene, or the development of the process in
eper tissues. There are the usual gastrointestinal symptoms of an
te fever. The tongue is coated, the breath foul; the urine scanty
d highly colored, with abundance of urates and frequently albumin.
in over the kidneys is frequent and the spleen may be enlarged. In
ne cases there is constipation, in others diarrhœa. Restlessness and
irium, most common at night, frequently occur, especially in old per-
s and when the disease is about the face and scalp.

Variations.—In many cases of erysipelas the process runs a very
ld course. The swelling of the involved area is slight, the color is
ldish, there is no formation of vesicles or bullæ, and upon subsidence
tle desquamation occurs (*erythematous erysipelas*). The fever does
t exceed 102° or 103° F. and the constitutional symptoms are corre-
ondingly moderate. Such an erythematous erysipelas may spread quite
idely, involving successively large areas of the body (*erysipelas migrans*).

In severe cases of erysipelas there is abundant formation of bullæ, and in rare grave cases the bullæ become hæmorrhagic. In some instances the process is so intense that gangrene occurs.

Occasionally, partly as the result of peculiar susceptibility, but chiefly in most instances because of the existence of some lesion, such as one in the nose, which is constantly exposed to infection, erysipelas recurs repeatedly during many years. In one case under my observation, a negro boy eighteen years old, there was a history of an average of two or three attacks of erysipelas per year for eight or ten years. In such cases permanent hypertrophy of the connective tissue may occur with the development of elephantiasis as the result of obstruction of the lymphatics. (Cf. illustration under elephantiasis.)

Complications.—It sometimes happens, especially when bullæ form upon erysipelas, that, as the result of infection with staphylococci, diffuse suppurative cellulitis supervenes. In such cases the swelling is increased, the tissues become boggy, then fluctuating, and unless evacuated the pus dissects its way along the connective-tissue planes and ulceration or gangrene occurs. When such a phlegmonous process supervenes upon erysipelas, drainage should be promptly provided.

During convalescence from erysipelas it is not infrequent for boils and superficial abscesses to appear.

Complications arising from the spread of the disease from the skin to the other structures are not uncommon. Involvement of the mucous membranes is the commonest complication of this kind. The disease may spread to the mucous membranes of the mouth, nose, pharynx, rectum, or vagina. When involving the fauces serious destruction of the tissues may occur from ulceration or gangrene, and in the larynx it is likely to produce obstructive œdema. When occurring about the genitals in females its spread to the uterus and adnexa produces a very grave complication. On the face and scalp not uncommon complications are meningitis and sinus thrombosis. It may spread into the eyes or ears and produce serious permanent destructive changes. It may extend through the Eustachian tube to the middle ear and pass thence through the external auditory canal to the scalp. From the mouth it has extended to the lungs and the œsophagus; is said in one case to have traveled entirely through the gastrointestinal tract.

It has long been noted from time to time that erysipelas sometimes acts as a curative agent in diseases in the course of which it occurs. Such improvement has been seen in various skin affections. The most remarkable effects have been in sarcomas and carcinomas. There are well-authenticated cases of sarcoma in which the disease has disappeared after severe attacks of erysipelas, and it was this fact that suggested the treatment of sarcoma with erysipelas toxins. Erysipelas has been observed also to benefit or cure severe neuralgia and to temporarily improve the mental condition of insane patients.

Etiology and Pathology.—Erysipelas is caused by streptococcus infection, and for its occurrence the presence of the organisms is necessary. The second essential factor is the presence of an abrasion. A contributory factor is lowered resistance of the tissues which renders them more vulnerable. The disease is most common in middle life. Certain individuals have an unusual susceptibility to erysipelas, and contract the disease almost whenever exposed to it. The predisposing causes are of the same character as those which predispose to furuncle and carbuncle. Among these must be mentioned especially chronic alcoholism. When this occurs in vagabonds, the combination of dirt and lowered resistance and frequency of slight wounds presents the ideal conditions for the development of erysipelas, and among such types the disease is especially frequent. Anders,[1] from an analysis of 2,000 cases, shows that erysipelas has rather curious seasonal variations. It is most frequent in the later months of winter and in early spring, and he finds a relationship to relatively low barometer and humidity. The probable explanation is that from February to April there is a relatively large proportion of persons whose resistance has been diminished by the trials of winter.

For infection with erysipelas a wound of the epidermis is necessary. It may develop in any sort of an abrasion, and trivial lesions, like slight cuts about the face and hands, abrasions in the nose, or pustules of acne or sycosis are often its starting point. Ulcerative processes in the nose and ears are frequently the points of infection, and in all sorts of persistent infected wounds its occurrence is not uncommon.

According to Fehleisen, erysipelas is produced by the growth in the cutaneous lymphatics of a specific streptococcus, but it is now generally believed that Fehleisen's streptococcus is identical with the streptococcus pyogenes. The streptococci are found free in the lymph spaces, in the connective tissue, and in the blebs, but they are not found in the blood or blood vessels except in rare cases of streptococcus septicæmia. They are most active in the spreading edge of erysipelas and rapidly disappear as the process subsides. Following Fehleisen, Miles[2] divides the distribution of the organism into zones, which are interesting as explaining the clinical phenomena that characterize the disease:

(1) In the spreading border just beyond the reddened area (the zone of greatest tenderness) the lymphatics are full of streptococci.

(2) Next behind this the tissues present all the evidences of acute

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[1] *Trans. Amer. Climatol. Assn.*, 1893–94.

[2] "Erysipelas," "Encyclopædia Medica."

inflammation with abundance of leucocytes which have taken up streptococci.

(3) Still further back the reaction is subsiding, the leucocytes are being destroyed by the fibroblasts or macrophages, and no free organisms are visible.

(4) Finally, when the process has ceased, the products of inflammation have been carried off and the tissues are normal.

While erysipelas in almost all cases is due to streptococcus infection, as Jordan[1] has shown, it may be produced by staphylococci. This observation has been confirmed by Pfahler and by Hajek.

Diagnosis.—The characteristic features of erysipelas are the angry, red, inflammatory swelling, the sharply defined borders, the shining tense surface, the tendency to peripheral spreading, the sudden onset, and the acute febrile disturbance accompanying it.

From the various forms of dermatitis which it resembles it is further distinguished by the character of its subjective symptoms. It burns and tingles and itches somewhat, but not so intensely as an acute eczema or dermatitis, and never sufficiently to cause scratching and the production of scratch marks.

Erysipelas may be confused with lymphangitis, phlebitis, acute suppurative cellulitis, acute spreading gangrene, and acute osteomyelitis. For detailed consideration of the differentiation from these surgical conditions, reference must be made to works on surgery. None of these affections should cause confusion except acute suppurative cellulitis. In this the clinical pictures may be very closely similar, but in suppurative cellulitis abscesses quickly form, the affected area becomes boggy, fluctuation appears, on incision free pus is found, upon the evacuation of which the temperature drops within a few hours almost to normal. The diagnosis from anthrax is considered under anthrax.

Prognosis.—The prognosis in erysipelas varies with the intensity of the process, the extent and the duration of the disease, and the general condition of the patient. In erysipelas of average intensity the prognosis is usually good. Even in severe uncomplicated erysipelas the outcome is more frequently good than would be expected from the severity of the constitutional symptoms. Death may occur from the intensity of the toxæmia, from exhaustion where the disease is severe and long continued, from invasion of deeper structures by the disease, or from complications in other organs.

Treatment.—An erysipelas patient should be isolated and the preventive measures taken which are indicated in other infectious diseases. There is danger of inoculation from a case of erysipelas as long as the process is active and until desquamation has ceased. After complete recovery the patient should have a general bath in bichloride of mercury (1:5,000) and his clothing and bedding should be sterilized.

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[1] *Münch. med. Wochenschr.*, 1901.

The internal treatment of erysipelas is limited to meeting therapeutic indications. The patient should have a generous but liquid diet. At the beginning he should have a sharp saline, or preferably a mercurial cathartic, and subsequently aperients to keep the bowels freely open. Where there is much depression alcoholic stimulants and strychnin are beneficial. Quinine has long been regarded as useful in erysipelas, and in moderate doses it is of benefit as a tonic and perhaps for some direct effect upon the sepsis. Tincture of chloride of iron is a classical remedy in erysipelas; it is probably not of any service, and as it is liable to upset the digestion it is better not given.

Marmorek's antistreptococcic serum has been used to some extent with favorable results by Marmorek, Robinson, Baum, and others as a specific agent to counteract the toxæmia of erysipelas. It is given in initial dose of about 20 c.c. injected into the abdominal wall and a dose of 10 c.c. is repeated every twelve hours until the temperature goes down. Following a personal recommendation of Dr. J. B. Murphy, I have used inunctions of ointment of colloidal silver in a few cases of streptococcus infection. A dram of the ointment can be rubbed daily into the healthy skin at any point. Murphy is of the opinion, after long experience with it, that it is useful in both streptococcic and staphylococcic infections.

The debility which often follows erysipelas needs tonics and supporting measures; change of scene and climate and rest are advisable for those who can avail themselves of these advantages.

The organisms in erysipelas are located so deep in the tissues that it is hopeless to attempt to destroy them by any external use of antiseptics. Their destruction must depend upon the inflammatory process which they excite, and the most that can be done in the way of meeting therapeutic indications is to prevent mixed infection, to allay irritation, and, perhaps, to prevent the spread of the process. Antiseptic precautions should be taken to prevent mixed infection; blebs should be opened, the surface kept clean, and soothing antiseptic applications should be used.

Wet dressings are a comfortable form of application. They may be used either hot or cool; hot compresses frequently changed are usually most satisfactory. Whether hot or cold, they should be changed frequently and not allowed to dry out. One of the most soothing applications is wet dressings of lead and opium wash. Other good applications are wet dressings of boric acid, of bichloride solution (1:5,000), of carbolic acid (one per cent), or of ichthyol (ten per cent). Alcohol is strongly bactericidal to the streptococcus of erysipelas, and frequent bathing of the parts with ninety-five per cent. alcohol is highly recommended and is not uncomfortable if the surface is not raw. This may be combined with wet dressings of carbolic acid (½ per cent in equal parts of alcohol and water), or of carbolic acid and resorcin (each ½ per cent in equal parts of alcohol and water). Ichthyol has been especially recommended in erysipelas, and it is a soothing application, either in aqueous solutions or in ointments, but it has no specific or extraordinary virtues.

Ointments or oils are preferable to wet dressings unless one can be sure that the wet dressings are changed so frequently that they do not become dry. Of the liniments carbolized oil (one half to one per cent) is one of the best soothing antiseptic applications. The ointments are made with the same drugs that are used in the wet dressings—ichthyol (ten to fifty per cent), carbolic acid (one per cent), boric acid (ten to fifteen per cent), resorcin (two to five per cent), and similar antiseptics in nonirritating strengths. Vaseline is the best base for ointments to be used in infectious processes. Where ointments are to be used they should be applied thickly spread on gauze, and the dressings should be changed before they become dry and stick to the surface.

It has long been suggested that the spread of erysipelas can at times be prevented by painting around the border with nitrate of silver or tincture of iodine, or some other irritating application. This has seemed so much like drawing a magic circle that it has been considered as a good deal of a form. But as Miles[1] has pointed out, by painting the skin just outside the border of the inflammatory process with tincture of iodine, or some other irritating application, an inflammatory process is excited, so that a large number of leucocytes are artificially brought to this location to act as phagocytes against the invading organisms. The method, therefore, has good scientific foundation. It is carried out by painting with tincture of iodine a line about half an inch wide around the affected area at a distance of about three quarters of an inch from the red border. Another way to prevent spreading which is well recommended is to paint around the border with plain (contractile) collodion. Carbolic-acid injections are also used for this purpose in the same way as in the treatment of carbuncle.

VIII.—VEGETATING LESIONS OF THE SKIN RESULTING FROM INFECTION

The supervention of hypertrophic tumorlike vegetations upon infected lesions of the skin is an occasional occurrence. This is seen most frequently in the lesions of syphilis called condylomata lata and in the nonsyphilitic lesions about the genitals called condylomata acuminata. In addition fungating masses of granulations covered by epidermis in which there is enormous thickening of the mucous layer are seen in rare instances in other dermatoses. As we have seen, vegetations occur upon the lesions of pemphigus in the form of the disease known as pemphigus vegetans, and their development has been observed also in rare cases of dermatitis herpetiformis. We also find these vegetations occurring in certain lesions of ringworm (kerion, agminate folliculitis, and some cases of tinea sycosis) and very rarely upon infected eczemas. Leaving out of consideration the vegetating lesions of pemphigus vegetans and of dermatitis herpetiformis, concerning whose character we are in no position to dogmatize, all of these conditions are characterized by infection and the exuberant formation of granulation tissue, and most of them by abundant suppuration. It seems highly probable that the vegetations in all of these conditions are an accidental complication resulting from a low grade of infectious inflammatory process, which produces lesions that are exactly analogous to the unhealthy granulations that have so long been known as proud flesh. The analogy to the formation of exuberant granulations by a simple inflammatory process is less accurate in the case of condylomata acuminata and condylomata lata than it is in the other conditions, but in these two conditions the mechanism of the production of the lesions is the same, and their occurrence can best be understood by bearing in mind the other vegetating lesions. It is quite possible, also, that the common papillary warts are of a very similar production, that they are the result of hyperplasia of the connective tissue and of the epithelium from some peculiar infection; but here we are venturing upon unknown ground, and the suggestion can be nothing more than an hypothesis.

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[1] *Edinburgh Hospital Reports*, vol. i.

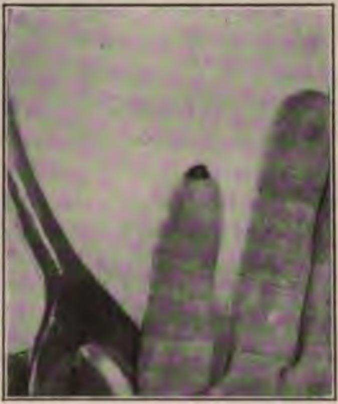

FIG. 125.—GRANULOMA PYOGENICUM. (Hartzell's collection.)

GRANULOMA PYOGENICUM

SYNONYM: *Botryomycosis hominis.*

Granuloma pyogenicum is the term applied to a small fungating tumor composed of granulation tissue which is the result of infection. The lesions are usually single, occur as tumors from the size of a small pea to an olive, are fungating or pedunculated, and of bright red color. They may be covered by a thin layer of horny epidermis, or may be denuded of epidermis. They are very vascular, bleed easily, and are usually quite tender to pressure.

The hypertrophic masses of granulations which sometimes develop upon vaccinations may be regarded as a fair picture of the condition.

They are persistent and tend to recur after removal. They occur, presumably always, upon the site of an abrasion or a wound, and they are usually seen upon the hands, because of the great liability to traumatism and infection of the parts, but they may occur upon any part of the body.

They were described by Poncet and Dor, who regarded them as specific neoplasms produced by botryomycetes. Subsequent investigations of Hartzell and others have shown that they are simply masses of hypertrophic granulations associated with infection with pus cocci. The lesions are highly vascular tumors with an abundant fibrous stroma and with great hyperplasia of the mucous layer of the epidermis. The organisms found in the pus and in the tissues are staphylococci.

They should be removed by thorough excision, which should be followed by light cauterization with caustic potash or chloride of zinc. Unless they are thoroughly removed and the last vestige of infected tissue got rid of they are likely to recur.

DERMATITIS VEGETANS[1]

SYNONYMS: *Dermatite pustuleuse chronique en foyers à progression excentrique*; *Pyodermatite végétante.*

Symptomatology.—Under the above names rare cases of inflammatory vegetating lesions of the skin have been reported. Most of the cases have supervened upon eczemas. The individual lesions are of the same type as those occurring in granuloma pyogenicum, and Wende has reported two cases in infants in which some of the lesions were discrete, pea-sized nodules like those of granuloma pyogenicum, and other lesions were large, approximating in size those in my case and others to be referred to immediately. In the cases which I have reported, and in similar cases reported by Hartzell, Hallopeau, and others, the fungating masses have been collected into large plaques. These plaques develop upon a weeping surface which has become infected, and consist of exuberant granulations which are piled up into fungating tumors. The masses are of bright- or dark-red color, may be covered by horny epidermis or denuded of it, and they have an abundant discharge of pus and serum with the consequent formation upon the surface of crusts. Where there is no weeping there may be more or less scaling.

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[1] Hartzell, *Jour. Cutan. and Gen.-Urin. Dis.*, 1901.—Wende, *ibid.*, 1902.—Perrin, *Ann. de Derm. et de Syph.*, 1900.—Hallopeau, *ibid.*, 1894.—Pusey, *Tr. Am. Derm. Assn.*, 1906.

mpanying these lesions there are usually areas of typical eczema ıer parts upon which vegetations have not occurred. The lesions ially apt to develop on the lower part of the abdomen and about als. In one of my cases, and also in Wende's cases, they occurred face, and in one of my cases they were found upon the arms, and the chest. The fungating masses are of variable size. In

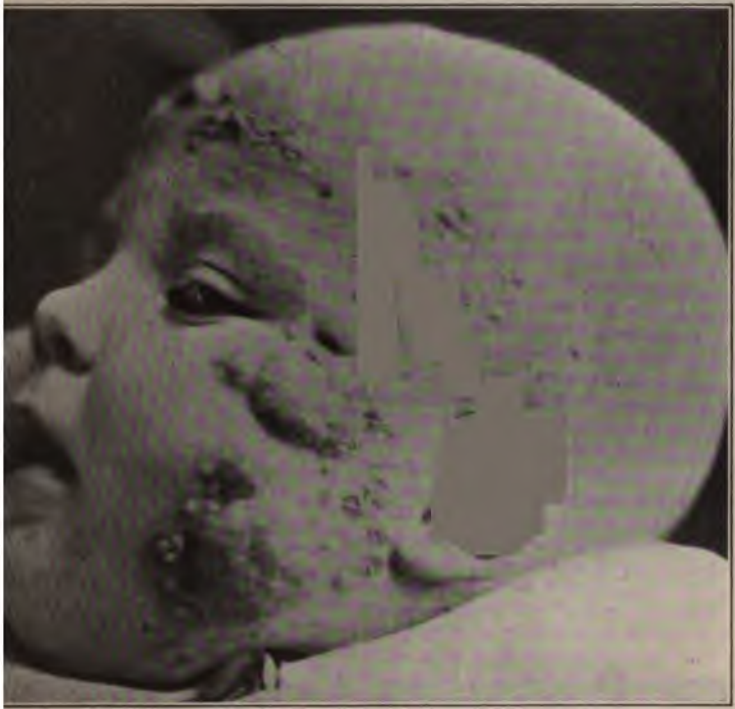

FIG. 126.—DERMATITIS VEGETANS, DEVELOPING UPON ECZEMA. (Grover W. Wende's collection.)

cases the masses varied from discrete tumors the size of a pea mors the size of a walnut. In most of the cases there have been s plaques upon the lower part of the abdomen, the inner sides ighs, and about the genitals. If they are left to themselves and s are not taken to check infection and suppuration, the lesions persistent, lasting for months or even years. With cleanliness septic precautions they subside within a few weeks, leaving temtains but no permanent scarring.

e is with these cases a varying amount of itching and of pain otion. Even the extensive cases are accompanied by practically itutional disturbance.

Etiology and Pathology.—The vegetating lesions develo eczemas which become infected. In many of the cases they have o in seborrhœic eczema. My cases occurred in patients who were u and lived under dirty conditions. It is probable that the veg occur chiefly in rating eczemas w neglected. The e factor for the tion of these ve plaques is appar inflammatory which becomes i and it is quite that these vege masses might upon other weep matoses besides Indeed, it is not that the vegeta pemphigus veget of dermatitis he mis are the sam secondary lesion

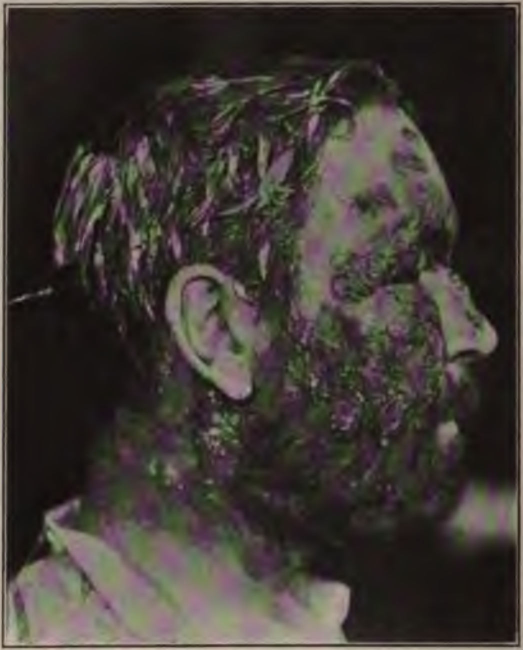

FIG. 127.—DERMATITIS VEGETANS, DEVELOPING ON PUSTULAR ECZEMA. (Author's collection.)

The conditio sentially the s granuloma pyog the lesions diffe size, number, a and extent of permanent connective-tissue formation. The lesic sist of masses of exuberant granulation tissue in a fibrous stro an abundant formation of blood vessels. The rete is greatly i in thickness with little increase in the horny epidermis. In the cases staphylococci have been found in the tissues and in usually the staphylococcus aureus.

Diagnosis.—The cases have to be differentiated from pe vegetans, dermatitis herpetiformis with vegetations, vegetatin lides, ringworm with vegetations, condylomata acuminata, blasto and mycosis fungoides.

In pemphigus vegetans there is a history of pemphigus; bull along with the vegetating lesions and are likely to be found mucous membranes; and there is increasing prostration an issue.

In dermatitis herpetiformis we have, as in pemphigus, the spontaneous eruption, apparently of neurotic origin, with polymor- lesions, usually vesicular or bullous, which occur in groups. The s are roughly bilateral in their distribution, appear and disappear ılarly in crops, and are accompanied by intense itching or other ctive disturbances.

ith vegetating syphilides there are always found other syphilitic s to discover the real nature of the disease.

esions similar to those of vegetating dermatitis might be produced ngworm. In such cases the diagnosis might depend altogether upon inding of the ringworm fungus.

he same is true of blastomycosis. In blastomycosis, however, the r is characteristic and is studded with pinpoint-sized abscesses from h the organism can be obtained, frequently in pure cultures.

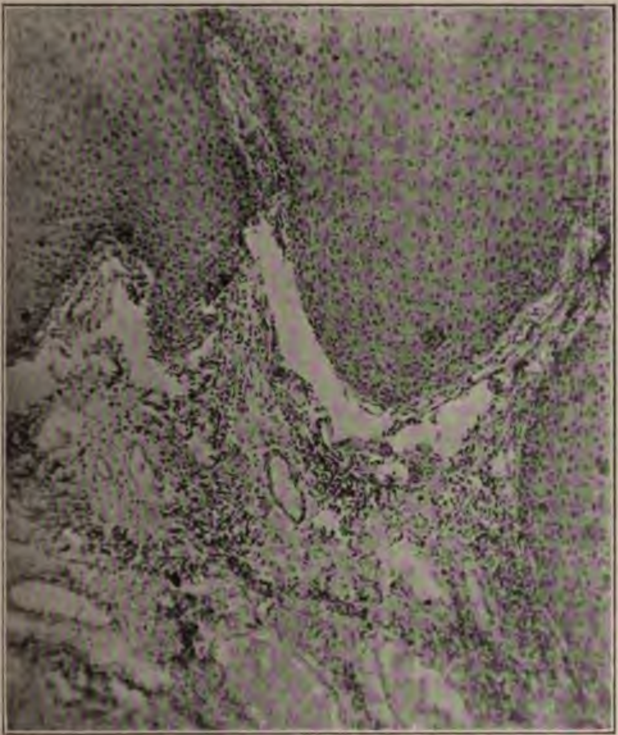

FIG. 128.—DERMATITIS VEGETANS. (Author's collection.)

ondylomata acuminata occur upon moist surfaces, especially about nuco-cutaneous junctures of the genitals and anus in association infection of these parts. The lesions are higher, more filiform in ture, and are usually few, but may form irregular masses as large as a hand. They are, however, never as extensive as the lesions shown about the genitals in the illustration given herewith.

The characteristic features of mycosis fungoides are clear enough to make a differentiation easy. In mycosis fungoides there is a history of a premycotic stage with the presence of sharply circumscribed patches of dry dermatitis, very persistent and very itchy. These are likely to be found in association with the later fungating masses of mycosis fungoides. The tumors of mycosis fungoides, while fungating, are roundish or lobulated, are dry in the early stage, and ulcerate late; they show no association with infection, and the natural tendency of the disease is to grow persistently worse. Histologically the tumors show the characteristics of sarcoid tumors.

Treatment.—The lesions yield readily to antiseptic treatment. In Wende's cases oleate of mercury ointment caused their disappearance in a few weeks. In the case which I treated the lesions rapidly disappeared when they were kept clean and dressed with boric-acid wet dressings.

CONDYLOMA ACUMINATUM [1]

SYNONYMS: *Verruca acuminata, Moist or Venereal Wart.*

Condylomata acuminata are vegetating masses which occur upon surfaces exposed to moisture and infection, especially upon the mucocutaneous junctures of the genitals, and consist of vegetating, wartlike growths from which there is an offensive discharge. They are not, like condylomata lata, lesions of syphilis.

Symptomatology.— They begin as small, filiform vegetations, which rapidly develop into tall, wartlike tumors. The lesions are quite sharply elevated, much more elevated and pointed than the syphilitic vegetations, and they are made up of groups of long, filiform vegetations, red in color, more or less denuded of epidermis, and covered with a grayish, foul exudate. There may be only one such lesion, perhaps of the diameter of a pea and a quarter of an inch high. More frequently the individual lesions of this size coalesce into irregular patches of the size of a coin or larger, and they may, in extreme instances, form a fungating mass entirely around the anus or entirely over the vulva. In such cases discrete condylomata will usually be found as satellite lesions. If these satellite lesions are beyond the area of maceration they may be covered completely with epidermis, form small, irregular, roundish or lobulated warty tumors, which look like a mass of hypertrophic granulations covered by healthy or horny epidermis.

Acuminate condylomata occur upon surfaces especially exposed to the effects of warmth, moisture, and infection, hence they are seen frequently and characteristically about the muco-cutaneous junctures of the genitals and anus. They also occur less frequently around the umbilicus, in the axillæ, under the mammæ, and around the mouth. If untreated they are very persistent, and as they are autoinoculable they are apt to extend in uncleanly persons. They cause no subjective disturbance except tenderness and pain upon movement.

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[1] Heidingsfeld, *Jour. Cutan. Dis.*, 1901, p. 226.

Etiology and Pathology.—The factors which favor their production are, as indicated above, warmth, moisture, and infection. They are usually, but by no means always, associated with suppurating conditions of the genitals and anus, and probably start in abrasions. It has not been determined that they are due to any specific organism, and it is probable that they may result from infections of different sorts. They are usually associated either with gonorrhœal infection or with infection with the common pyogenic organisms. It is probable that their pathogenesis is essentially the same as that of the lesions of vegetating dermatitis just considered.

Histologically there is inflammatory hyperplasia of the corium, especially of the papillary layer and abnormal proliferation and hyperplasia of the mucous layer of the epidermis.

Diagnosis.—They are most likely to be confused with condylomata lata. Syphilitic condylomata occur upon the same sites, but they are flat vegetations with a papillary surface, but without the filiform projections and prominent elevation of acuminate condylomata. Syphilitic condylomata arise from syphilitic papules, and such papules can nearly always be found in association with condylomata lata. In addition, the history of, or evidence of, recent secondary syphilis can always be obtained. Acuminate condylomata are usually regarded as a form of common digitate or filiform warts. That this is not the case is, in my opinion, indicated by several facts: by their invariable occurrence upon surfaces showing abundance of moisture and warmth; their association with infection, usually with suppurative processes; and their inflammatory character. Not infrequently discrete filiform warts do appear upon the genitals. In such cases the lesions are of a less acute inflammatory type, are often single, and tend to remain dry without suppuration.

Treatment.—The best treatment of condyloma acuminatum is removal by excision. After being clipped off, the base should be lightly cauterized. Large masses may be treated by the constant application of antiseptic wet dressings. For this purpose, because of its deodorizing and astringent qualities, potassium permanganate (1:2,000 or 1:3,000) is a good application. Under antiseptic dressings the lesions shrink and may entirely disappear, but frequently their destruction is necessary. Simple cauterization with nitrate of silver does no good.

IX.—CHANCROID[1]

SYNONYMS: *Soft Chancre, Simple or Noninfecting Chancre.*

Symptomatology.—Chancroid is an autoinoculable ulcer occurring about the genitals which develops within two or three days after infection and runs an irregular and uncertain course. At the site of inoculation of chancroid there develops within six to twelve hours an inflammatory papule which within twenty-four to forty-eight hours is converted into a pustule upon an inflammatory base. This ruptures, leaving a small ulcer which, by peripheral extension behind an inflammatory border, increases to the size of a finger nail, or larger. The ulcer thus produced has a base of unhealthy grayish granulations, over which there forms a yellowish necrotic pellicle. The borders of the ulcer are undermined and have a minute ragged edge, best observable with a hand glass. There is a free secretion of pus which is usually mixed with more or less blood. The ulcer is situated upon a base of inflammatory œdema. This shows moderate inflammatory induration but not, unless cauterized, the dense, buttonlike induration of chancre. Ordinarily the lesions vary in size from a pea to a finger nail. They are usually rounded or oval, but may be quite ragged and irregular. When numerous adjacent lesions coalesce, large irregular ulcers may be formed, and occasionally the lesions show a widely spreading tendency, become phagedenic, and develop into extensive and serious ulcers. There may be only one ulcer in a case of chancroid, but as the pus is autoinoculable the ulcers are frequently multiple and may be numerous. One lesion offers no protection against the development of others, so that in a case of multiple chancroid, lesions may be found in different stages of their course.

Chancroids occur about the genitals and on the skin of the adjacent parts. They are very rarely seen in other locations; when this occurs it is usually by autoinoculation from genital lesions. Upon the mucous membranes of the genitals they are more destructive in their course than upon the skin, and when occurring under a long prepuce they may be followed by destructive ulceration through the prepuce. In chancroids under these circumstances there is a violent inflammatory reaction, usually with the production of phimosis or paraphimosis.

Chancroid appears within two to three days after inoculation and its duration depends upon circumstances. It may last only ten days or two weeks, or, in unfavorable cases, may persist for many weeks before the infection is stamped out and healing brought about.

Within a few days after the appearance of chancroid on the genitals, it is not uncommon for the inguinal glands to become affected, usually on one side, but frequently on both. As a result of this complication there occurs an infective adenitis with the production first of a painful, tender node beneath the skin; then the inflammatory process spreads until it involves the skin, and if suppuration occurs (which is the usual outcome) an abscess is formed over which sooner or later the skin breaks down unless incision is previously made. Occasionally this does not occur, and the adenitis subsides from the acute inflammatory stage. This glandular enlargement is likely not to occur if pains are taken to keep the ulcer clean and prevent the formation of crusts. If the ulcer becomes crusted so that drainage is not free, a bubo is very apt to be formed. Along with adenitis there is frequently painful lymphangitis, and occasionally an abscess forms in the course of the dorsal lymphatics of the penis (bubonulus).

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[1] Chancroid is illustrated with chancre.

Complications.—In addition to suppurating bubo other complications are lymphangitis, phimosis, paraphimosis, phagedena, and gangrene. These are all surgical complications for detailed consideration of which reference must be made to works on surgery or venereal diseases. Phagedena and gangrene are now exceedingly uncommon.

Etiology and Pathology.—Chancroid is usually produced by inoculation from another chancroid and is usually of venereal origin, but this is not always the case. The affection occurs most frequently in the careless and dirty.

Whether chancroid is a specific ulceration produced by a specific organism or is a lesion due to the common pyogenic organisms is a question concerning which there is room for difference of opinion. A chancroidal ulcer has no clinical characteristics which can distinguish it from an ulcer due to infection with common pyogenic cocci. In its clinical appearance the ulcer is indistinguishable from an ecthymatous ulcer, such as may be produced by pus organisms upon (say) the thighs or buttocks. On the other hand, the common pyogenic organisms are always found around the genitals and very uncommonly produce ulcers simulating chancroidal ulcers, while infection from a chancroidal ulcer usually reproduces itself as a chancroid. This may be explained by supposing that the chancroidal ulcer is produced by pyogenic cocci of unusual virulence. A majority of authorities, perhaps, do not accept this view, but regard chancroid as a specific ulcer, and Ducrey has found an organism in the pus and Unna has found the same organism in the tissues of chancroid which they regard as the specific organism of the disease. The Ducrey-Unna bacillus has not been grown in artificial media, so that its pathogenic character remains open to question. The bacillus is a short and thick, dumb-bell-shaped organism with rounded ends and a slight constriction in the middle. It is a streptobacillus which grows usually in parallel chains. It is found both outside and within the cells; it has not been found in the pus from buboes. It stains readily with fuchsin or gentian violet and is decolorized by Gram.

Diagnosis.—The diagnosis of chancroid is exceedingly important because upon it frequently rests the question of syphilis. The characteristic features of chancroid are its rapid development within one to three days after inoculation; its rather destructive ulcerative tendencies; the frequent involvement of the adjacent glands in a suppurative inflammatory process; its autoinoculability and the frequency of multiple lesions; the absence of induration of the base beyond simple inflammatory induration; and its comparatively painful course.

Chancroid has to be diagnosticated from chancre, mucous patches, ulcerating gumma, herpes of the genitals, epithelioma, and perhaps at times from suppurating lesions of the genitals due to scabies.

In the chancre there is a definite and comparatively long period of incubation, from two to four or five weeks; there is a base of well-marked induration which feels like a sharply circumscribed button in the skin, as hard as paraffin; the surface is eroded rather than ulcerated, and ulceration, if present, is slight. The lesion as a whole is insignificant, and runs a sluggish, painless course, and to the uninitiated is apparently of trivial character. The chancre is not autoinoculable; it is usually single, but if infection has occurred in several points multiple lesions may develop at the same time; if they do they all run the same course. The involvement of the contiguous glands in syphilis is practically invariable, but is a noninflammatory, painless, hard enlargement, quite different from that of chancroid. In a typical chancre an expert can make a practically sure diagnosis, but in chancroid it is never possible to say that there is not a mixed infection, so that in chancroid it is never possible to positively exclude syphilis until a sufficient period has elapsed for secondaries to manifest themselves.

Syphilitic mucous patches about the genitals may be confused with chancroids, particularly if they ulcerate from secondary infection. A typical mucous patch is a superficial erosion, sharply circumscribed and covered by a grayish pellicle, which cannot readily be confused with a chancroid. If a confusing infected lesion should develop upon a mucous patch, the presence of typical mucous patches about the genitals or in the mouth, and the presence of other secondary manifestations of syphilis in the skin and glands should render the diagnosis easy.

Ulcerating syphilitic gummata about the genitals are deep, excavated, destructive ulcers which appear as deep nodules that melt away. They are unhealthy, sluggish, comparatively painless, and without inflammatory involvement of the adjacent glands. A previous history of syphilis should be obtainable, and they heal only upon the application of specific treatment.

The diagnosis from herpes of the genitals has been considered under herpes.

Epithelioma of the genitals appears as a nodule which ultimately breaks down, is of slower development, has characteristic nodular borders, is painful, is likely to occur in later life, and is especially associated with some long-continued source of irritation, such as phimosis. In doubtful cases the examination of a section from the border determines the true character.

Ulcerating lesions from scabies about the genitals will be associated with scratch marks and the presence of scabies lesions between the fingers or on other parts of the body.

Treatment.—Patients with chancroid should have as complete rest as possible; they should certainly avoid unnecessary exercise, and are best off in bed. Where the patients are reduced from disease or debauchery, they should be placed under as favorable circumstances as possible, and need the supporting treatment indicated generally in infectious conditions occurring in such individuals.

The local treatment may be carried out by providing surgical cleanliness or by taking measures to convert the lesion into a noninfected granulating ulcer. If the first course is pursued the ulcers should be kept scrupulously clean, and in order to minimize the chances of bubo the formation of crusts should be carefully avoided. The ulcers should be cleaned at least twice a day with an antiseptic solution applied as hot as can comfortably be borne, such as solution of bichloride (1: 1,000 or 1:2,000), of potassium permanganate (1: 500 or 1: 1,000), of carbolic acid or of lysol (one or two per cent). These irrigations should be carried out with an abundance of solution and each application should be continued for several minutes. After irrigating it is a good plan to have the parts immersed in hot water or hot boric-acid solution for half an hour or more; in gangrenous or phagedenic cases the continuous bath of this sort for many hours in the day—six to twelve hours in the twenty-four—is the best form of treatment. After irrigating it is customary to dry the ulcer and apply an antiseptic powder, such as aristol, calomel, or iodoform. The objection to these is that they form crusts and do not take up the secretion, and a better plan is to dress the ulcer with a boric-acid or weak carbolic-acid compress.

The second method, which has for its aim the destruction of the infected area and the conversion of the lesion into a noninfected ulcer, is carried out by the use of destructive caustics. The following is the most satisfactory method of carrying out this procedure. First irrigate the ulcer carefully with antiseptic solution. Then paint it well with a five- to ten-per-cent solution of cocaine. After anæsthesia is p dry it carefully; this should be done thoroughly in order to pre running of the acid to be applied subsequently. After thoroug ing touch the entire surface of the ulcer, both edges and bas careful to let no points escape, with pure carbolic acid, applied or of cotton on the end of a toothpick. If the infection is superf painting with carbolic acid may be sufficient to destroy it, but i cases it is not enough, so that to be safe it is necessary to appl acid after the application of the carbolic. This is done lightly b oughly over the entire surface, and, like the application of carbo it must reach every point of the ulcer, otherwise reinfection wi at once. Immediately after cauterization the ulcer should be with sterile water to stop the action of the acid, and should dressed with cold compresses of boric-acid solution or simila solution. After this procedure there is an acute inflammatory r the slough is thrown off, and a healthy granulating wound is le advantage of this radical method of treatment is that, when it thoroughly carried out and the patient subsequently watched to reinfection, healing takes place much more quickly and the da bubo is greatly reduced. It should not be undertaken unless t ditions for carrying out the treatment are favorable.

No caustic treatment of chancroid should be undertaken u is radical. For this reason cauterization with nitrate of silver cially to be avoided. It simply sears over the surface, does not the infective process, and, a larly objectionable feature, p an intense inflammatory in which cannot readily be distin from that of syphilis.

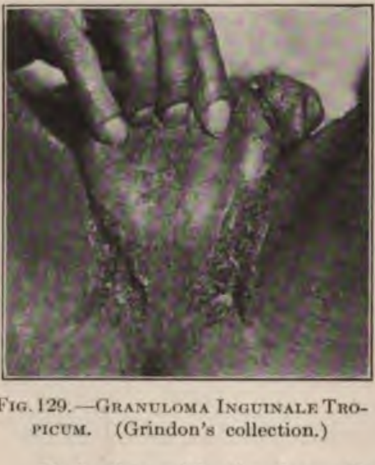

FIG. 129.—GRANULOMA INGUINALE TROPICUM. (Grindon's collection.)

GRANULOMA INGUINALE TRO

SYNONYMS: *Groin Ulcer*, *U Granuloma of the Pudenda* (G

Under this name are d linear ulcers, with raised p borders usually in the groin, are seen chiefly in tropical co

The disease is apparently common in the West Indies.

It begins with the formation of nodules or tubercles in t which break down and coalesce into large ulcers. The lesions r

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[1] Galloway, *Brit. Jour. Derm.*, vol. ix, 1897.

are linear in form, usually following the line of the groin, are perhaps an inch wide and several inches long, with an elevated papillary border and more or less papillary overgrowth of the surface. Ulceration and healing may alternate, so that in a lesion ulcerations will be found intermingled with areas of scar tissue. The disease is very chronic, lasting from a few weeks to several years.

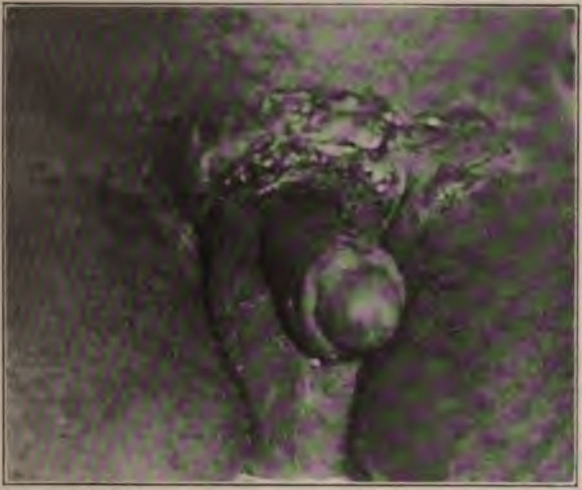

FIG. 129a.—GRANULOMA INGUINALE TROPICUM. (Grindon's collection.)

It is said to be contagious. Crocker regards it as probably due to pus infection, and has seen similar linear papillary ulcerating lesions in the groin in England. Crocker recommends curettement of the lesions and the application of carbolic acid.

X.—ANIMAL POISONS

The pathogenic and saphrophytic bacteria which flourish in dead animal matter are an abundant source of infectious processes in the skin. These are usually forms of infection with the pyogenic staphylococci or streptococci which pursue the usual course of such infections, producing suppurative cellulitis, lymphangitis, and erysipelas. Not infrequently anthrax and other specific infectious diseases are transferred to man in this way, as they are from living animals. In addition there is a certain number of indefinite, probably infectious, inflammatory processes which occur in persons who handle animal matter which has undergone more or less decomposition, like meats, hides, fish, or cadavers. Most of these conditions present fairly definite clinical pictures, but the pathogenic organisms have not as a rule been determined. There seems little reason to doubt that they are usually one of the pyogenic staphylococci or the streptococcus pyogenes. The typical conditions of this sort are erysipeloid, post-mortem pustule, and gayle. Erysipeloid is the most characteristic.

ERYSIPELOID [1]

SYNONYMS: *Erythema migrans, Erythema serpens, Crab Cellulitis.*

Erysipeloid is an affection of the skin which occurs from poisoning by dead animal matter through abrasions in the skin, and is characterized by the occurrence of a patch or patches of slowly spreading dermatitis with little or no constitutional reaction.

The condition is probably not uncommon in persons who handle dead animal matter, but it does not come frequently to the dermatologist's attention. It was first clearly described by Morrant Baker as erythema serpens, but its recognition has only become general since it was thoroughly studied by Rosenbach and given the very pat name of erysipeloid.

Symptomatology.—The disease begins at the point of entrance of the poison as a dry, red or purplish patch, slightly swollen and tense, and with a sharply defined border. It spreads slowly at the periphery and clears up behind. It may appear at several points, which spread until they coalesce and form circinate patches. The color varies from reddish to dusky red, and the swelling, ordinarily slight, may at times be considerable. The disease occurs characteristically on the fingers and about the joints and knuckles, although Elliot has recorded a case in which it was transferred from the fingers to the toes, and Gilchrist has reported among his 329 cases, 2 on the soles. The disease, as a rule, does not spread widely; beginning on one finger or a knuckle, it may spread from one finger to another until several fingers or a considerable part of the hand is involved. In rare cases it may spread to some extent up the forearm. There is very rarely any involvement of the lymphatics or contiguous glands; in Gilchrist's 329 cases, 5 showed enlargement of the epitrochlear or epitrochlear and axillary glands and 3 lymphangitis.

There is some burning and itching of the involved area. The parts are stiff and tender and hot, and pain may be considerable. The constitutional disturbance is practically nil. There may be slight malaise and febrile disturbance, but ordinarily the constitutional symptoms are so slight as to be overlooked and the temperature remains normal.

The incubation period is usually about two days, but may be as short as three to twelve hours. The disease persists from three or four days two or three weeks; occasionally where it spreads more widely, longer. n the end it disappears spontaneously without desquamation.

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[1] Fordyce, *Jour. Cut. and Gen.-Urin. Dis.*, 1896.—Baker, Morrant, *St. Bartholomew's Hospital Reports*, vol. ix, 1873.—Rosenbach, *Verhandl. der Deutsch. Gesellschaft f. Chirurgie*, vol. xvi, 1887.—Elliot, *Jour. Cut. and Gen.-Urin. Dis.*, 1888.—Gilchrist, *Jour. Cut. Dis.*, 1904.

Etiology and Pathology.—The disease is caused by poisoning from eats, fish, poultry, cheese, and similar animal products. It has arisen rom dissecting wounds. Gilchrist has recorded 329 cases in Baltimore; f these, 323 were produced by crab bites or from lesions caused by ard crabs; of the other 6 cases, 4 were from fish bones, 1 was from eat, and 1 was from pigs' feet. The affection is seen chiefly in those hose occupations involve handling animal food products, and in such ersons recurrences are not uncommon. The poison is always introuced through an abrasion.

Rosenbach believed that the disease was due to a specific microöranism, which developed in decomposing meats and belonged to the mily of cladothrix. Gilchrist's very thorough bacteriological studies ave discovered no definite organism. There can be little doubt, hower, that it is an infectious process.

Diagnosis.—Its etiological and clinical features are sufficiently aracteristic to leave little room for difficulty in diagnosis. From ysipelas it is distinguished by the milder character of the local reacn and the almost entire absence of constitutional symptoms; from gworm by the absence of the vesiculo-papular or scaly border of that ndition and of ringworm fungi; from eczema by its origin, like an fectious process, in an abrasion, its serpiginous course and sharply deed border, and by the tenderness and pain and the relative absence of hing which characterize it.

Treatment.—The disease yields readily to antiseptic applications. lchrist recommends the application of twenty-five-per-cent salicylicd plaster, which should be applied firmly and well beyond the border the lesion. One application of this plaster for three days is often fficient for cure, applying subsequently for a few days a simple bland tment to the sodden epidermis. It may be treated with other antitic ointments or wet dressings, such as are used in the treatment of sipelas.

DERMATITIS REPENS[1]

SYNONYMS: *Acrodermatitis perstans, Acrodermatite suppurative continué.*

Dermatitis repens is a dermatitis which appears as the result of uries on the upper extremities, and is characterized by a persistent dency to spread.

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[1] Crocker, p. 235.—Hartzell, *Jour. A. M. A.*, December 20, 1902 (full bibliohy).—Hallopeau, *Annales*, 1897, "Acrodermatites continués"; *Revue Générale Clinique et de Thérapeutique*, February 12, 1896, Hallopeau and Leredde—Stowers, t. *Jour. Derm.*, vol. viii, 1898.—Audry, *Annales de Derm. et de Syph.*, vol. ii, 1 (full bibliography).

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d successfully by different authorities. Whatever the method of
t used, it has to be carried out persistently.

POST-MORTEM PUSTULE

ortem pustule is a superficial ulcer produced by infection from
It appears at the site of an abrasion as a red, inflamed, pain-
which develops into a pustule upon an inflammatory base.
ries into a crust upon the surface and the lesion may extend
by ulceration. Other lesions may result from inoculation.
t of the lymphatics and contiguous glands is not uncommon,
las and suppurative cellulitis may develop from the lesions.
anism of post-mortem pustule is not definitely known, but
from the description of this lesion that it presents no essen-
ces from similar lesions produced by infection with the
genic organisms. It is probably not a specific disease, and
as a clinical entity is a relic of antebacteriological days.
produced by infection from a cadaver should be opened
pon its appearance and thoroughly cleaned; the base should
th pure carbolic acid and then with alcohol, after which
of 1:1,000 bichloride solution should be applied. If
d if a crust is not allowed to form over the lesion, there
pt healing. If a crust is allowed to form and remain,
ction has not free exit, the bacteria are apt to make

It is a rare disease. It was first described by Crocker,[1] who regards it as a neuritic affection, but it is placed here because of its resemblance to erysipeloid and because its objective symptoms strongly suggest infection.

Symptomatology.—The disease begins with the appearance of vesicles or bullæ at the site of an injury, which may be slight. The vesicles rupture with exfoliation of their covering, and leave a bright red surface from which a clear or turbid serum exudes. The borders of the lesions thus formed are sharp, and are marked by a fringe of sodden epidermis which is thrown up by the undermining exudate. The disease spreads at the border and tends to heal at the center. Fresh vesicles and bullæ may form adjacent to the original lesion, and coalesce with it. The disease is practically always unilateral. Ordinarily it does not extend beyond the hand, but Crocker has seen it spread up to the elbow, and, in a very persistent case, even up the entire arm, across the back, and down the other arm to the elbow. The condition is very persistent, and may last, in the widely spreading cases, for months or even for a year or more.

Dry Form.—Crocker has seen three cases which presented the characteristic features of the disease except that the lesions appeared as dry, inflammatory patches that spread peripherally and did not at any time in their course show any free exudate upon the surface.

Acrodermatitis Perstans.—Under the name acrodermatitis perstans Hallopeau has described a condition closely analogous to, if not identical with, dermatitis repens. Features which it presents differing from dermatitis repens are the facts that constant recurrence may take place for many years; in some cases the disease is spread by the development of distant fresh foci, and large areas of disease have been produced by the coalescence of such foci, while in dermatitis repens the disease only increases by continuous extension. In two cases of acrodermatitis perstans, furthermore, the mucous membrane of the mouth has been involved, and in one case a fatal impetigo herpetiformis developed.

Etiology and Pathology.—The disease starts as a result of an injury, often quite slight. Apparently basing his opinion upon its persistent course and relative intractability to treatment, Crocker regards it as due to peripheral neuritis, but suggests that its spread is caused by secondary parasitic infection. Hallopeau regards both his and Crocker's forms as of microbic origin, and the staphylococcus albus has been found in many cases. It seems hard to understand how peripheral neuritis could cause a lesion to spread continuously, as happened in one of Crocker's cases, up one arm, across the back, and down the other arm, and the character of the lesions and the fact that they ultimately yield to antiseptic applications strongly suggest that they are of bacterial origin.

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[1] *Brit. Med. Jour.*, December 11, 1888.

Diagnosis.—The disease has to be differentiated from erysipeloid and eczema. The differences from erysipeloid have been referred to under that condition. From eczema and various forms of dermatitis due to external irritation it is distinguished by its origin in a wound, its sharply defined, undermined border, and its extension by continuity.

Prognosis.—The cases of dermatitis repens all recover ultimately, but some are very rebellious to treatment. Acrodermatitis perstans shows a persistent tendency to recurrence, and the cases are of long duration.

Treatment.—Mild soothing antiseptic applications are not sufficient in the treatment of these conditions. Crocker recommends that the undermined epidermis at the border be carefully cut away and the affected area painted daily with a ten-per-cent solution of potassium permanganate for a week. As a result of this a black crust forms, which separates in a few days. Iodoform rubbed into the surface or applied on dressings is sometimes successful. In the dry form, Crocker had success by applying salicylic acid and creosote plaster to the edge until the scaly border could be removed, and then using compresses of bichloride solution 1:4,000 until the surface became irritated, after which boric-acid ointment was used. Various antiseptic dressings have been used successfully by different authorities. Whatever the method of treatment used, it has to be carried out persistently.

Post-mortem Pustule

Post-mortem pustule is a superficial ulcer produced by infection from cadavers. It appears at the site of an abrasion as a red, inflamed, painful papule which develops into a pustule upon an inflammatory base. The pus dries into a crust upon the surface and the lesion may extend peripherally by ulceration. Other lesions may result from inoculation. Involvement of the lymphatics and contiguous glands is not uncommon, and erysipelas and suppurative cellulitis may develop from the lesions.

The organism of post-mortem pustule is not definitely known, but it is evident from the description of this lesion that it presents no essential differences from similar lesions produced by infection with the common pyogenic organisms. It is probably not a specific disease, and its separation as a clinical entity is a relic of antebacteriological days.

A pustule produced by infection from a cadaver should be opened immediately upon its appearance and thoroughly cleaned; the base should be swabbed with pure carbolic acid and then with alcohol, after which a wet dressing of 1:1,000 bichloride solution should be applied. If this is done, and if a crust is not allowed to form over the lesion, there is usually prompt healing. If a crust is allowed to form and remain, so that the secretion has not free exit, the bacteria are apt to make their way into the lymphatics and produce a serious deep infectious process. When this supervenes it is, of course, treated as under other circumstances.

VERRUCA NECROGENICA

SYNONYMS: ***Post-Mortem Wart, Anatomical Tubercle, Tuberculosis verrucosus cutis.***

Verrucæ necrogenicæ, or post-mortem warts, are a form of lupus which develop about the knuckles as result of inoculation of tubercle bacilli from cadavers. They are described under lupus verrucosus.

GAYLE[1]

Gayle[2] is a grave form of puerperal fever which occurs in sheep. The organism producing this disease is sometimes inoculated upon the hands of men who have to do with the dead animals, and it gives rise to a superficial lesion in the skin which, in contrast with the fatal septicæmia in sheep, runs a very benign course.

At the point of infection there develops upon an inflammatory base a vesicle with clear or blood-stained serous contents. This enlarges peripherally until there is formed a lesion, perhaps an inch in diameter, of bluish-gray color, and with a slight red areola. Accompanying the lesion there may be considerable swelling of the hand, and the axillary glands are usually enlarged. Pain and constitutional symptoms are absent or very slight.

Klein has shown that the disease is due to a specific organism which he calls staphylococcus hæmorrhagicus.

The disease yields readily to mild antiseptic applications. The vesicles should be ruptured, the surface cleansed, and an antiseptic wet dressing, like bichloride 1:2,000, applied.

XI.—MALIGNANT PUSTULE[3]

SYNONYMS: ***Anthrax, Pustula maligna, Wool-sorter's Disease.***

Malignant pustule is a specific carbunclelike lesion of the skin produced by the bacillus anthracis, the infection usually having its origin in animals suffering from splenic fever. Anthrax is a disease of the horse and donkey, and to a less extent of some other animals, from which it may be communicated to man. It is an uncommon disease in man.

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[1] Crocker, p. 509; Klein, *Brit. Med. Jour.*, August 4, 1897.

[2] Not to be confused with *gale*, the French for Scabies.

[3] Woodhead, Sims, "Bacteria and Their Products."—Delépine, "Splenic Fever," *Encycl. Medica.*—Bell, Anthrax, Allbutt's "System of Medicine."—Koranyi, Article "Der Milzbrand," *Nothnagel's Specielle Pathologie und Therapie*, vol. v, Wien, 1900 (full bibliography).

Symptomatology.—The development of malignant pustule is usually divided into four stages: first, stage of incubation, one to three days; second, stage of formation of the vesicle, twenty-four to thirty-six hours; third, stage of development of inflammation, induration, and œdema, one or two days; fourth, stage of gangrene and sloughing of the lesion. Beginning with the penetration of the organism in an abrasion in the skin, there is for one to three days no evidence of the disease. Then at the site of infection an inflammatory papule begins to develop, accompanied by itching and tingling; in the course of twenty-four to thirty-six hours the papule develops into a vesicle or bulla with clear or blood-stained serous contents, or a pustule on an angry red inflammatory base. The bacilli are then abundant in the exudate and for some distance around the lesion. The bulla or pustule ruptures, and there forms beneath it a brownish or black gangrenous slough the size of a small coin, sur-

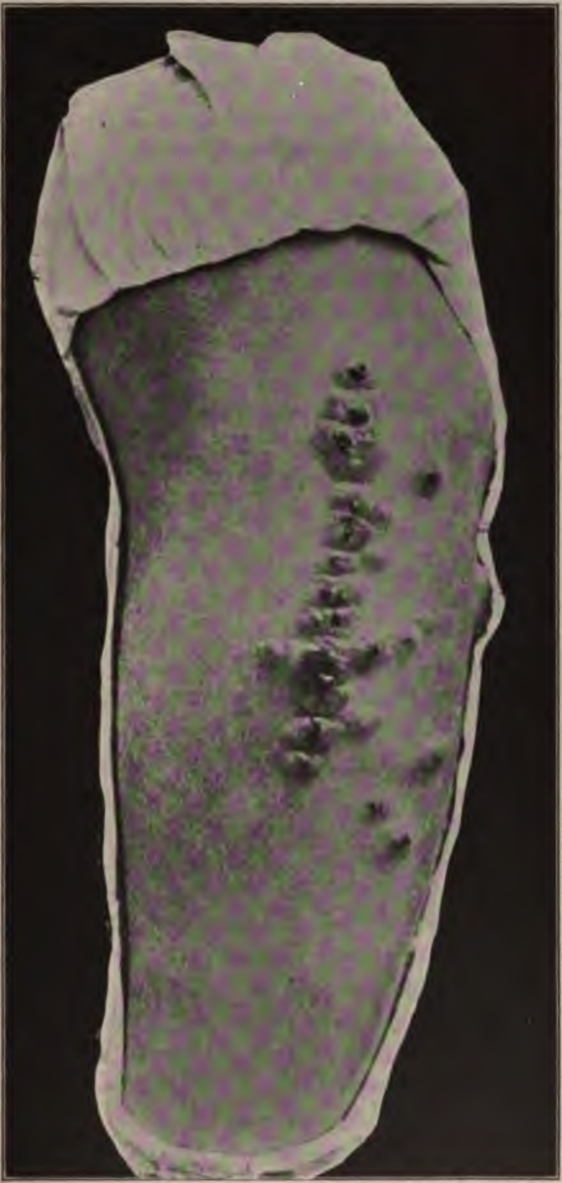

FIG. 130.—ANTHRAX. (Reproduction in black and white from Jacobi's dermochromes. By permission of The Rebman Company.)

rounded by a border of vesicles or pustules. At the same time the inflammatory induration around the lesion greatly increases; the skin is densely infiltrated, swollen, tense, of dusky red color, with a sharply defined border, around which the tissues are intensely œdematous, forming a pale or pink areola, and the contiguous glands and lymphatics radiating from the lesion are inflamed and swollen. At this stage the bacilli are most abundant at the border of the slough and in the surrounding lymphatic spaces. The slough at this stage is from one or two to five or six centimeters in diameter. It may not increase beyond this size, or after its primary development may spread more widely. After reaching its full size, if the patient has not succumbed to general sepsis, the slough separates, leaving a deep ulcer which heals by granulation.

Usually the lesions are single, but when infection occurs at more than one point two or more malignant pustules may develop simultaneously. As would be expected, the lesions occur upon exposed parts. Virchow found that in eighty-four per cent of the cases they were upon the face, neck, hands, fingers, or forearms.

As long as the disease remains local the constitutional symptoms are those of a toxæmia. Human tissues are fairly resistant to anthrax bacilli, so that infection not infrequently remains local. During the course of malignant pustule, however, the organisms may at any time gain entrance to the blood, and in that event they produce a quickly fatal anthrax septicæmia, and this accident is about as liable to happen with small lesions as with large ones. When the disease becomes general there are all the evidences of a violent septic infection: rigors, sweats, temperature of 104° F. or more, severe pains in the head and bones, typhoid state, coma, and death in from thirty to forty hours up to, in less severe cases, four to six days.

Etiology and Pathology.—The organism of anthrax grows readily outside of living tissues; may, indeed, as Koch demonstrated, live for years as a saphrophyte, so that infection may occur directly from animals or persons, or indirectly from materials upon which the organisms exist. Infection may occur from handling hair and hides or such materials months and even years after the organism has been deposited upon them. It may be transmitted by the bite of flies and other insects which have fed upon the blood or tissues of diseased animals, and in one case it has been transmitted by the bite of a dog that had recently fed upon an animal dead of the disease.

In malignant pustule infection takes place through the skin. Infection may take place through inhalation of the organism or through its ingestion; with such infections there develops primarily an anthrax septicæmia.

One attack of anthrax generally confers immunity in animals, and presumably in man.

The discovery and the study of the anthrax bacillus is one of the early and important facts in the history of the bacteriology of infectious diseases. Because of the large size of the organism and the readiness with which it grows outside of the body it was the first of the pathogenic bacteria to be thoroughly investigated, and its study has been of the utmost importance in the development of our knowledge of pathogenic bacteria. The bacillus was recognized in the blood and the spleen pulp of cows as early as 1849 by Pollender, although his observation was not published until several years later, and in the next year it was discovered by Davaine and Rayer. In 1863, stimulated by Pasteur's studies upon the relationship between microörganisms and butyric fermentation, Davaine demonstrated that the organism was the cause of splenic fever and anthrax. The only link lacking in his chain of evidence was the obtaining of pure cultures of the organism outside of the body and the reproduction of the disease from these, facts that were not demonstrable in the state of knowledge at that time. This final demonstration was made by Koch in 1876.

The anthrax bacillus is a large, rod-shaped, nonmotile organism of an average diameter of 1.2 microns and an average length of 5 to 6 microns. It is readily grown on most media, and stains easily. In general anthrax the organism can be found in the blood, in the tissues, in the fluid from the external lesions, in the sweat, sputum, urine, and fæces. The bacilli are easily destroyed but the spores are very resistant, surviving prolonged immersion in five-per-cent carbolic-acid solution and resisting boiling for three or four minutes. Boiling for about thirty minutes is necessary to insure their destruction.

Diagnosis.—The diagnostic features of the disease are the occupation of the patient, the presence of a rapidly developing gangrenous lesion with a border of vesicles surrounded by an œdematous collar, and the frequent violent constitutional symptoms. The diagnosis must be confirmed immediately by discovering the organism, which can readily be done by staining smears. The lesions resemble carbuncles, especially those on the face. Their more rapid course and the rapid appearance of gangrene serve to distinguish them clinically.

Prognosis.—About thirty-three per cent of cases of malignant pustule die. According to Hamer's statistics of cases in London forty per cent of those upon the neck died, twelve per cent upon other parts of the body. In other statistics fifty per cent have died.

Anthrax septicæmia is exceedingly fatal. In sixty-three cases observed by Bell, forty-four cases died between the second and fourth days, and only two survived over ten days.

Treatment.—If the lesion is seen in time it should be thoroughly excised, the excision extending well beyond the area invaded by the organism, and this should be followed by the use of irrigations and wet dressings of 1:20 carbolic-acid solution or 1:1,000 bichloride. Thorough destruction by the actual cautery has long been a method used in countries where the disease is common, and is well worthy of consideration, for in excision there is always the possibility of not getting beyond the field of growth of the bacteria or of spreading them with the knife.

XII.—EQUINIA[1]

SYNONYMS: *Glanders, Farcy.*

Equinia is a specific infectious disease which is caused by the bacillus mallei and is characterized by the formation of nodules, especially in the mucous membranes of the nose, in the skin, and in the subcutaneous lymphatics, which tend to break down with the formation of destructive ulcers.

Symptomatology.—The lesions of glanders are infective granulomata; those occurring in the skin do not differ from those in the mucous membranes, but the clinical picture of the disease varies according as the lesions in the nose predominate (glanders) or those beneath the skin (farcy), so the division of the disease into glanders and farcy is of clinical convenience.

Acute Glanders.—After a short prodromal febrile disturbance there appears a tenacious nasal discharge—first mucous, but quickly becoming purulent, bloody, and abundant. This discharge is the result of the development of lesions in the nose, and as they break down destructive ulceration occurs. This not only involves the soft parts of the nares and the contiguous tissues, but may affect the bones. There is great swelling of the face, the skin over the nose is tense and red, the conjunctivæ are injected, the eyelids swollen, perhaps to the point of closing the eyes, and the glands of the neck much enlarged. As the disease becomes well established there appear on the surface, especially of the face and limbs, groups of red macules which quickly become yellow-tipped papules, increase to the size of a pea, and become pustules on a livid red base, thus producing an eruption which is a good deal like that of variola. As the groups of papulo-pustules break down they form irregular sloughing ulcers or gangrenous patches. Along with these lesions the subcutaneous lymphatics, not only adjacent to the original infection but in various parts of the body, become enlarged, and deep-seated nodules appear upon them (farcy buds). Some of these may undergo involution, while others break down into deep, irregular, foul ulcers.

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[1] Nuttall and Pigg, Article "Glanders," *Encyclo. Medica.*—Councilman, Article "Glanders," Wood's "Reference Handbook of the Medical Sciences."—Woodhead, Article "Glanders," Allbutt's "System of Medicine."

Acute Farcy.—When infection occurs through the skin there may develop, first, a group of dark-red papules, which quickly become pustules and break down into unhealthy, spreading ulcers, or the process may begin as a phlegmonous cellulitis; the affected area becomes greatly swollen, tense, red, and painful, and breaks down into a foul, spreading, irregular ulcer with undermined edges and dirty, sanious discharge. This phlegmonous cellulitis may spread until it involves an entire limb. Along with these manifestations at the site of inoculation in the skin the papulo-pustular lesions and farcy buds appear upon the general surface.

Thus, in both the glanders and farcy type of the disease the lesions are widely distributed and very abundant, and form a hideous picture of a destructive generalized infectious process in the skin.

Constitutional Symptoms.—In connection with the infection in the mucous membranes or in the skin there is general infection with the organism and frequent development of lesions in the deep structures of the body, with the constitutional symptoms of a grave pyæmia.

There is ordinarily a short incubation period of three to four days; occasionally it is as much as a week, and possibly as long as three weeks. The appearance of the local lesions is preceded for a short time by moderate febrile and rheumatic prodromal symptoms; these increase as the disease develops; the temperature goes up to 103° to 105° F. or more; severe rigors and sweats occur; the articular and muscular pains increase and become distressing, and the patient passes into a typhoid state and death occurs.

Chronic Glanders.—In chronic glanders and farcy the course of the disease may be so masked as to present little similarity to the acute form. In the glanders type there is chronic nasal discharge, with more or less destructive ulceration of the nares, and perhaps of the larynx. In such cases a diagnosis may be possible only by bacteriological tests.

In the farcy type the nodular tumors occur especially about the extremities. They break down, forming deep, sluggish ulcers with little inflammatory reaction and without marked involvement of the glands.

In both chronic glanders and farcy the constitutional symptoms are minimized, and may be absent. The disease may persist for months, and even years, the patient finally recovering, or dying from the development of pyæmia or acute glanders.

The glanders organism may lodge in deep-seated tissues and produce a localized focus of the disease without the development of its characteristic clinical picture, as illustrated by a case reported by Tadeschi of chronic osteomyelitis produced by the bacillus mallei, in which death occurred from glanders meningitis.

Etiology and Pathology.—Glanders occurs chiefly in the horse, mule, and donkey, other animals showing varying susceptibility to it. It is a rare disease in man, and practically always results from infection, either direct or indirect, from the horse. The organism is found in the discharge from the nose and the ulcers, in breaking-down tissues, pus, urine, saliva, and milk, and the disease may be transferred not only directly from one animal to another, but indirectly through organisms which have found lodgment upon all sorts of articles. Infection usually occurs through the respiratory mucous membrane or through abrasions of the skin. It may occur from infected food, through the gastrointestinal mucous membrane, and Babes and, later, Cornil and Nocard infected guinea pigs through the hair follicles of the uninjured skin by applying the bacilli in vaseline; so that infection through the intact skin is a possibility.

There is a certain variation in the susceptibility of different individuals and in the virulence of the organism under different circumstances which probably accounts for the differences in the intensity of the clinical manifestations.

The lesions are granulomatous tumors, in which the bacilli can be found. The organism is a short, nonmotile bacillus of somewhat variable form, but similar to the tubercle bacillus. It stains readily, and can be grown easily on potato or blood serum. Outside of living tissues, the bacillus mallei is not the persistent organism that the anthrax bacillus is. Aërobic cultures lose their virulence in a few days, and under the most favorable conditions of culture they lose their virulence in four months (Löffler). Exposed on threads to sunlight, they are destroyed in one day. They are readily destroyed by antiseptics (by 1 : 2,000 bichloride in one hour, by five-per-cent carbolic acid in five minutes).

Diagnosis.—In acute cases the clinical features are sufficiently characteristic after the cutaneous or nasal symptoms develop to render the diagnosis clear, but the diagnosis should always be confirmed by discovery of the organism. Before the development of the characteristic lesions in the skin and nose, the disease may be mistaken for rheumatism or typhoid fever. In the chronic cases the diagnosis is made by finding the organism, by the mallein test, and by inoculation experiments. Following the injection of mallein there is a violent reaction at the site of injection, causing great swelling, but if done aseptically there is never suppuration. There is also a characteristic acute constitutional reaction similar to that following the tuberculin test. This test is a routine procedure in the care of horses where the disease is suspected.

Strauss's inoculation test may be carried out as follows: cultures from the secretions are made upon agar-agar, and injections of this are made into the peritoneal cavity of a male guinea pig. At the end of two to three days, if the case is glanders, the scrotum becomes reddened and the testicles swollen. They continue to swell and finally suppurate, and death occurs in two or three weeks from generalized glanders.

Prognosis.—Both glanders and farcy occur in acute and chronic forms. The acute forms are invariably fatal, usually within eight to ten days. The chronic forms may drag on for months and years, and recovery takes place in about one half of the cases, although acute glanders or pyæmia may supervene upon the chronic form and prove quickly fatal.

Treatment.—In the management of a case every precaution should be taken to prevent spread of the disease. This involves isolation of the patient, great precaution to avoid direct infection of attendants, and destruction of all materials coming in contact with the patient or their thorough sterilization by boiling or by soaking in two-and-one-half-per-cent carbolic-acid solution. (One-half-per-cent carbolic acid kills the organism in two to four hours, Bonhoff.)

If seen in the beginning, the original lesion should be excised or thoroughly destroyed by caustics. Buds should be opened before they break down and ulcers dressed antiseptically. The internal treatment is symptomatic, and is devoted to supporting the patient in every way possible and in carrying out the usual measures indicated in the treatment of septicæmia. Mallein has been claimed to be of some value as a therapeutic measure. Mallein, however, like tuberculin, is of doubtful benefit as a therapeutic agent, and is chiefly valuable for diagnostic purposes in horses.

XIII.—ORIENTAL BOIL[1]

SYNONYMS: *Aleppo Boil, Biskra Button, Delhi Boil, Kandahar Sore, Gassa Button, Pendjeh Sore, Natal Sore* (?).

Aleppo boil is an infectious ulcer pursuing an indolent course which is endemic in certain tropical localities. It was first described in 1845 by Peacock, who saw it in Aleppo.

It begins as an inflammatory papule which has been compared to an inflamed mosquito bite. This gradually increases in size and forms a tense, glistening, dark-red, sluggish tubercle, the surface of which is intact but is studded, as can be seen with the hand glass, with minute yellowish points, presumably abscesses. In the course of a few weeks to several months the nodule breaks down with the discharge of a serous or sero-purulent fluid, which dries upon the surface in a brownish crust. Beneath this crust ulceration takes place and gradually extends, until there is formed a round ulcer from three quarters of an inch to two inches in diameter. It is sluggish, with sharp and irregular undermined edges and with a base covered by unhealthy, fungoid granulations and secreting thin, offensive pus. The ulcer occurs characteristically upon the face, next most frequently upon the hands and feet, but may occur upon almost any part of the body, although it is not described upon the scalp. The lesions, usually single, may be multiple, and are usually discrete; occasionally they become confluent, forming then irregular, large ulcers. They last from two to twelve months or longer, but ultimately heal with the formation of puckered, pigmented scars. Glandular involvement is not a characteristic feature, but the lesions are apt to be complicated by infection, with the production of erysipelas, lymphangitis, and adenitis. There is no constitutional disturbance.

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[1] Murray, J., *Trans. Epidem. Soc.*, vol. ii, 1883.—Wright, *Jour. Cutan. Dis.*, vol. xxii, p. 1, 1904.—Strong, "Some Tropical Ulcerations of the Skin," *Philippine Jour. of Science*, vol. i, No. 1, January, 1906.

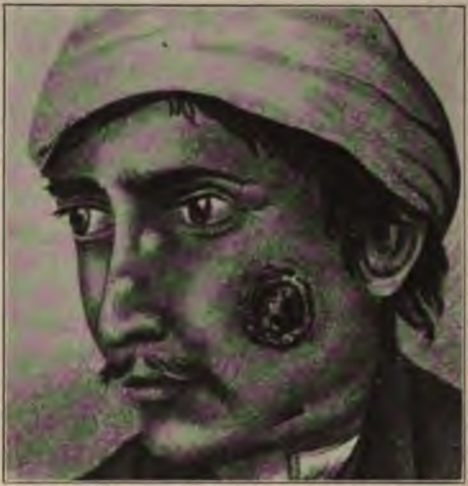

FIG. 131.—ORIENTAL BOIL. (Photographic reproduction from Kaposi's Handatlas der Hautkrankheiten.)

Etiology and Pathology.—The disease is endemic in various tropical countries, as indicated by its numerous names. It is infectious, and is transmissible both to man and to animals. It is generally considered to be a specific infection, but the pathogenic organism has not been determined. Wright believes the disease is due to a protozoön which he has found. He regards the organism as the same as the one described by Riehl, and probably the same as that described by Cunningham and by Firth. The organism is thought to occur in water in infected localities, and inoculation from this source is regarded as the common origin of the disease.

Diagnosis.—The characteristic features of the disease are its limitation to endemic localities, its slow, indolent, and benign course, its predilection for the face, and the absence of constitutional symptoms.

Treatment.—The lesions heal promptly upon excision, curettement, r destruction by vigorous caustics. After any of these radical proedures the wounds are treated according to ordinary surgical priniples. Finsen light has been used in one case successfully, and Crocker uggests as probably a better method of treatment the use of x-rays.

XIV.—VERRUGA PERUANA[1]

SYNONYMS: *Peruvian Wart, Carrion's Disease, Oroya Fever.*

Verruga peruana (Spanish, *verruga,* a wart) is a disease endemic in certain valleys of the Peruvian Andes which is characterized by a prodromal febrile disturbance of several weeks' duration, with the subsequent development of wartlike granulomatous tumors upon the skin and in other structures.

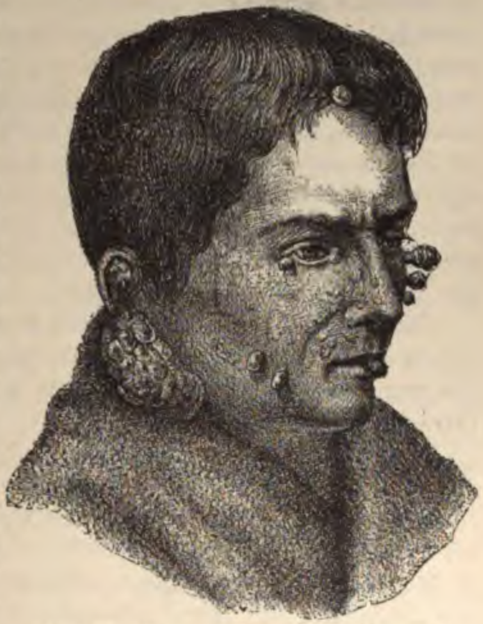

FIG. 132.—VERRUGA PERUANA. (Dounon.)

Symptomatology.—The disease may be divided into an incubation period, a febrile period, and an eruptive period. The incubation period, which is without definite symptoms, may vary from eight to forty days; in the case of Carrion, symptoms began to develop twenty-two days after experimental inoculation. The disease in the febrile period has the characteristics of a severe acute infectious fever which it may be impossible to distinguish from acute articular rheumatism, severe malaria, or typhoid. The temperature may rise to 104° F., rarely higher. The most characteristic features of this period are distressing muscular pains and cramps, pains in the bones and joints, and the rapid development of extreme anæmia. The severity of the febrile disturbance varies greatly in different cases; in very severe forms of the disease—Oroya fever—patients frequently die in the febrile stage before the appearance of the eruption.

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[1] Matas, Morrow's "System," Article "Verruga."—Beaumanoir, *Arch. de Méd. Navale Coloniale,* Jan., 1891 (abstract in *Archive de Derm. et de Syph.,* vol. ii, 1891).

After a febrile disturbance of uncertain duration, which is said to vary from twenty-eight days to six or eight months, or even more, the eruption begins to appear. With its appearance there is usually very great improvement in the constitutional symptoms. The anæmia, however, persists and is apt to be made worse by the hæmorrhages which frequently occur from the tumors. The lesions appear, first upon the face and extremities, then over the body, as reddish spots or inflammatory vesicles or pustules, which subsequently develop into the characteristic tumors. These are highly vascular granulation tissue tumors which vary in size from a pea to an orange. They may be conical, rounded, or pedunculated, are soft and elastic and tender, and covered by smooth, shining epidermis. The smaller tumors, after reaching their height of development, may gradually shrink, dry up, and fall off or be absorbed. The larger tumors, either as a result of scratching or without traumatism, frequently slough and form unhealthy, sluggish ulcers. With the sloughing of the tumors there is frequently severe hemorrhage, which increases the anæmia and often leads to death. In addition to these cutaneous lesions, subcutaneous lesions occur, chiefly on the extremities, as round, movable tumors; at first they do not involve the skin, but ultimately they are likely to spread to the skin, break down, and form unhealthy, deep ulcers. There is not usually any lymphatic enlargement.

The number of lesions may vary from a few discrete lesions on the extremities and face to a very abundant eruption. Even when abundant, the trunk is usually comparatively free. Not only the skin and mucous membranes are involved in the lesions, but they have been found also in many of the internal organs.

The course and duration of the eruptive period is variable. The lesions do not usually occur all at one time, but appear irregularly, and the eruptive period may persist from a few days or weeks to two or three months or more.

The eruption is accompanied by itching sufficiently severe to cause the patient to scratch and wound the lesions. The constitutional disturbances of the eruptive period are those arising from cachexia and extreme anæmia.

Etiology and Pathology.—The disease has remarkable geographical limitations. It is endemic exclusively on the Pacific slope of the Peruvian Andes, and there only in definite districts, showing a predilection for certain valleys from 700 to 2,600 meters above the sea level (Dounon). In these localities it has evidently existed since the earliest historical times. It is mentioned as early as 1543 by Zárate, in his history of Peru.

It is undoubtedly a distinct disease, having pathognomonic characteristics. It occurs in all races which have been exposed to it, and not only in man, but in horses, mules, asses, cattle, hogs, and dogs. It affects both sexes and all ages, and is especially severe in children. Daniel E. Carrion, a medical student, apparently proved, in an experiment that cost his life, that the disease is inoculable. The method of transmission of the disease is unknown. It is locally believed to be contracted from the water in the infected districts. It has occurred in most malignant forms in association with railroad building and the upturning of immense amounts of earth in the endemic districts. It is possible that its sharp restriction to certain localities is explained by the fact that it is transmitted by some insect whose habitat is only these districts.

The lesions have the structure of infective granulomata, so that the disease presents some analogies to leprosy and syphilis. The tumors are composed of highly vascular granulation tissue, and in large lesions show a structure at the center almost like that of cavernous angiomata.

V. Yzquierdo, of Santiago, Chili, who has studied the disease very thoroughly, has discovered in the lesions a large bacillus similar to but larger than the tubercle bacillus. It is found not only in the tissues of the tumors, but in the blood vessels, where it occurs in such abundance as at times to cause their occlusion. While the Yzquierdo bacillus falls short of the complete demonstration of its pathogenic character, there is strong reason to believe it to be the pathogenic organism.

Prognosis.—The disease is an exceedingly grave affection, having a fatality of eight to ten per cent in the most favorable groups of cases, and reaching a fatality in the famous epidemic accompanying the building of the Oroya railway of sixty to seventy per cent. The prognosis is better the milder the constitutional symptoms and the earlier the development of the eruption.

Treatment.—The treatment is symptomatic. The most important matter is to remove the patient from the district, preferably to the seashore, as quickly as possible after the discovery of symptoms. This measure is said to have an exceedingly beneficial effect upon the disease.

XV.—YAWS[1]

SYNONYMS: *Frambœsia, or Pian* (West Indies), *Buba* (Brazil), *Coko* (Fiji), *Tonga* (New Caledonia), *Bouton d'Amboine* (Moluccas), *Patek* (Sumatra), *Dubea, Ajortor*, or *Tongara* (Gold Coast), *Ogodo* (Sierra Leone), *Effier* (Benin), *Soombah, Gattoo, Framosi, Tetia*, or *Momba* (various West African names), *Kwena* (Burmah), *Lupani* (Samoa), *Purru* (Malay Peninsula).

Yaws is a specific, infectious disease, occurring in tropical countries, which is characterized by more or less constitutional disturbance and by the development in the skin of reddish tubercles which are capped by a cheesy crust.

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[1] Clayton, Article "Yaws," *Encycl. Med.*—Nicholls, "Twentieth Century Practice of Medicine."—Powell, *Brit. Jour. Derm.*, 1896.—Daniels, *ibid.*, 1896.—Finu-

The term yaws is applied not only to the disease as a whole, but also to the cutaneous lesions which characterize it.

Yaws has long been known and has a large literature. It was first mentioned by Oviedo in 1535, and was described by Pison in 1648. The extent of its distribution is suggested by the numerous names given above. At the present time its existence is notable in tropical Africa, especially on the west coast, and in the West Indies, although it is distributed widely throughout the tropical world. There are some differences in details of description among different writers—differences, however, which throw no doubt upon the characteristic features of the clinical picture of the disease—and in the description given here I follow closely the very clear and logical account of the disease given by Clayton, who has studied it upon the west coast of Africa.

Symptomatology.—The disease has an incubation, a febrile, and an eruptive period. The incubation period varies from two to ten weeks; it probably averages about four weeks. In the inoculation experiments of Charlouis it averaged about fourteen days; in those of Paulet, twelve to twenty days. Acquired in the usual ways, the inoculation period is generally believed to be longer. During this period there are no definite symptoms. The febrile period begins with a febrile attack lasting from one or two days to a week or more. This may be so slight as to pass unnoticed or it may be severe, and is characterized by fever, with its usual accompanying symptoms. Deep-seated rheumatic pains, even when the constitutional disturbance is very slight, are characteristic. During the febrile period there is usually some general enlargement of the lymphatic glands, especially of those near the site of inoculation. Each successive outbreak of the eruption may be preceded by such a prodromal constitutional disturbance.

The febrile disturbance is followed by the appearance of the eruption, which begins as a papule at the site of inoculation, or if the inoculation has occurred in an already existing sore, this develops into a small ulcer. The primary lesion of yaws at the site of inoculation suggests the initial lesion of syphilis. It, however, does not differ from later yaws, and is typical of the lesions which occur throughout the course of the disease. In this respect, of course, it presents striking contrast to the initial lesion of syphilis. Accompanying the development of the original lesion there appear irregular-shaped, scattered patches of branny desquamation. In these scaling patches minute pap-

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cane, *Jour. Trop. Med.*, April, 1901.—Rat, "Yaws, Its Nature," 1891.—Hutchinson, *Archives of Surgery*, vols. vii and ix.—Hirsch's "Geographical Pathology."—MacLeod, *Brit. Med. Jour.*, September, 1901.—Henggeler, *Monatshefte*, March 1, 1906.

ules soon begin to develop, and "gradually push their way through the epidermis, which splits over them and curls away from their summits." There then appears upon the tips of these papules a yellow

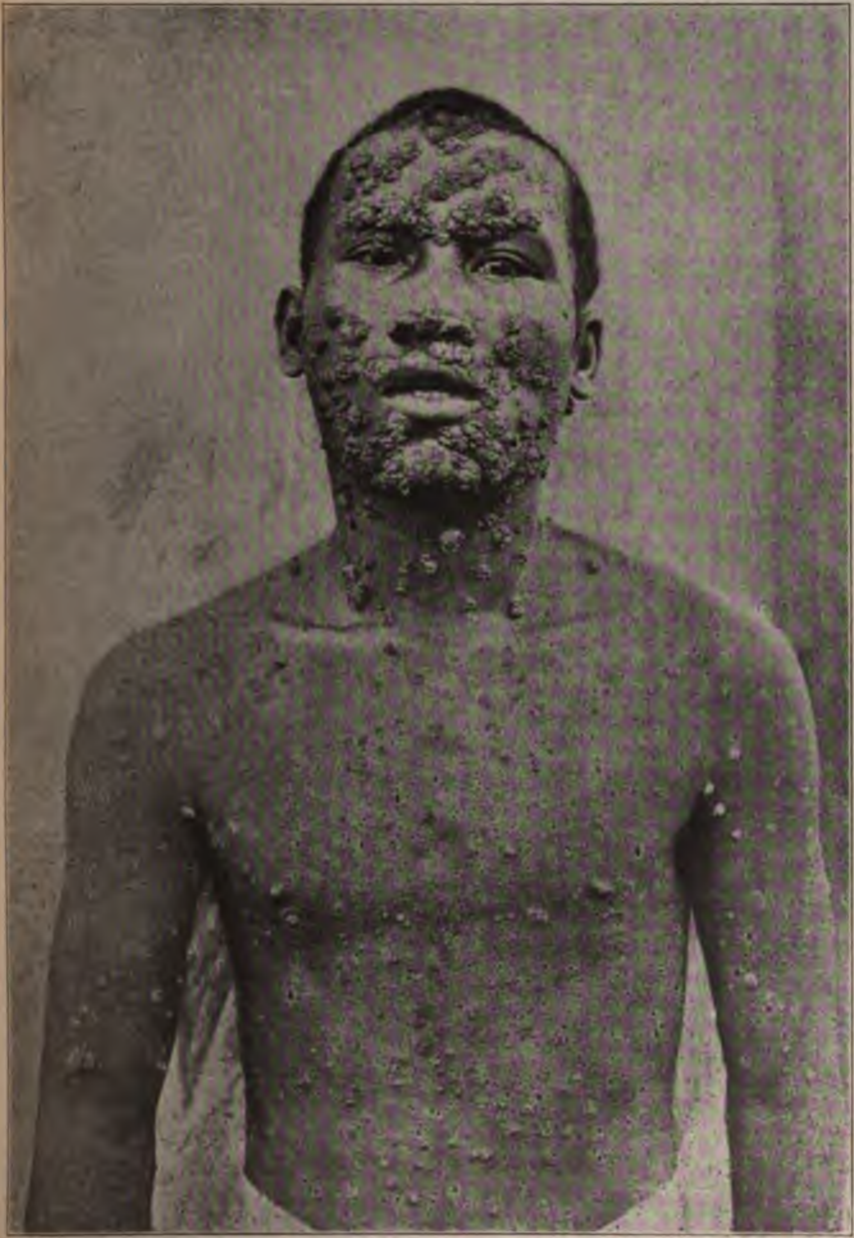

FIG. 133.—YAWS. (Henggeler.)

discharge, which dries into a characteristic cheesy crust that has been regarded as a sebaceous secretion, but apparently cannot be such. The lesions may not pass beyond this papular stage, but may persist as such, and ultimately subside. This papular type is a very persistent form of the disease. From the papular stage, however, the lesions usually develop into tubercles from the size of a pea to a walnut or larger, rounded or flattened in shape, and capped by the characteristic cheesy crusts. Adjacent lesions coalesce into large irregular tumors, especially about the mouth and nostrils, or may develop with a circular arrangement and coalesce into circular figures with a healthy center (*ringworm yaws*). Where the lesions are exposed to moisture, as about the mouth and nostrils and anus, they become fungoid, weeping, and sodden, with a very offensive discharge, and resemble the flat condylomata of syphilis. Frequently one yaw increases rapidly in size, and becomes markedly larger than the other lesions (*mother yaw*), and is apt to be very persistent. The removal from the lesion of the cheesy crust, which is usually closely adherent, exposes gray or red granulations which, from oozing of blood from the papillæ, will be dotted with minute blood crusts. Immediately upon removal of the crust there occurs an outpouring of secretion and the rapid re-formation of the cheesy cap.

The lesions vary greatly in number as well as in size. They may be localized or widely distributed. They affect by predilection the face and extremities; the trunk is comparatively free and the scalp usually not involved. Frequently yaws appear under the nails, and produce troublesome onychia. Lesions occur upon the muco-cutaneous junctures of the nose, mouth, and anus, but they do not ordinarily occur upon the mucous membranes. In certain districts, as Fiji, however, they are described in the nasal mucous membrane and upon the soft palate, where they are accompanied by destructive ulceration; but these locations are atypical, and very uncommon. Yaws do not normally ulcerate, but as a result of traumatism and infection with pyogenic organisms ulceration is a not uncommon complication, occurring in about eight per cent of the cases (Nicholls). As a result of infection extensive ulcers may develop and produce deforming scars. The larger lesions which occur upon the soles, on account of the pressure to which they are exposed and on account of the thick epidermis which binds them down, form irregular, flattened tumors which undermine the epidermis and finally rupture, then become infected, and leave fungoid, fetid, ulcerating, tumors.

The lesions reach their development in about two weeks, remain stationary for a short time, then begin to shrink, the crusts dry and fall off, and the lesions finally disappear, leaving pigmented spots. The shrinking of the lesions takes about four weeks. In unhealthy subjects the lesions may persist much longer. Where ulcers occur they usually heal readily under treatment. The eruptive period lasts from weeks to years, the disease continuing itself in prolonged cases by successive crops of lesions. As the eruption appears in crops, it is usual to find at one time in a given case the early scaly patches, papules, and typical yaws or nodular lesions. In the course of an intercurrent acute disease the eruption may disappear, as a rule, however, only temporarily.

During the development of the eruption there is considerable itching. The lesions themselves are not only not tender, but are insensitive, so that the crust can be forcibly removed without pain. The West Indian negroes are said to distinguish the lesions of yaws by applying lime juice to them to see if they are insensitive. A peculiar musty odor, which becomes acrid and offensive in severe cases, characterizes the eruption.

During the period of eruption there may be febrile attacks preceding outbreaks of the disease, or there may be no acute disturbance of the health. Anæmia, more or less intense, is, however, a constant symptom of the eruptive period, and there may be pronounced cachexia.

Etiology and Pathology.—Yaws is a disease of tropical countries, and occurs chiefly among those who live in tropical filth and dirt. Whites are largely immune from the disease. This may be due to their more careful habits of living, but there may be also to some extent a natural immunity. It occurs in both sexes and may occur at any age, but the greatest liability to the disease is in childhood, between five and fifteen. The disease is never congenital, and there is no evidence of hereditary transmission.

That it is inoculable has been proved by the experiments of Charlouis and Paulet. It is apparently communicated only through abrasions in the skin. The poison remains active outside of living tissues, and can be transmitted through clothing and similar articles, and seems to persist in huts. Flies are regarded in endemic localities as carriers of the infection.

There is a difference of opinion as to whether one attack protects against another.

The histology has been studied by various observers, recently by MacLeod. The lesions show the structure of granulomatous tumors with no tendency to permanent organization. There is excessive proliferation of the rete with parakeratosis of the horny layer and downgrowth of the interpapillary processes so marked that the structure resembles that of condylomata acuminata.

Diagnosis.—The characteristic features of yaws are: the formation of indolent, fungoid nodules covered by peculiar cheesy, closely adherent crusts; the painless character of the lesions; the tendency to heal spontaneously without scarring; and the occurrence of the disease in natives in endemic localities.

Castellani in 1905 discovered a spirochæta (spirochæta pertenuis) in yaws, whose invariable presence in the disease has been confirmed by many other observers. Morphologically it is very similar to the spirochæta pallida. Russell, from comparative study of the two organisms, distinguishes the following differences between the spirochæta pertenuis and the spirochæta pallida: the spirochæta pertenuis is slightly thicker; the distance from crest to crest of the spirals is greater; as a rule, the distance from the crest to the hollow of the spirals is greater; the spirals are not quite so regular in their height. Prowasek has noted the same differences. Neisser, Baermann, and Halberstädter, in an experimental research on yaws, came to the following conclusions: Yaws can be transmitted from man to the higher and lower apes; and it can be transmitted from ape to ape. There is a diffusion of its poison throughout the whole body. Animals infected with syphilis can be successfully inoculated with yaws. Castellani has obtained a complement fixation reaction in the disease.

Prognosis.—The disease tends to spontaneous recovery. There is some mortality (2.5 per cent, Nicholls), from intercurrent infections in ulcerating lesions or from diseases to which the patients are rendered especially susceptible through cachexia.

Treatment.—Much can be done to prevent the development of the disease by attention to cleanliness and to proper hygienic conditions. Where segregation has been systematically carried out, as in the Barbadoes, the disease has practically disappeared.

The patients should be put under proper hygienic conditions, and built up with an abundance of nourishing, digestible food. The debility and anæmia should be treated with tonics, among which arsenic, quinine, and strychnine are especially recommended. The mercurials and iodides are not useful. From the favorable effect of intercurrent, acute febrile disturbances upon the disease, the use of thyroid extract and erysipelas toxins has been suggested, in order to produce an artificial fever.

The local lesions, where they become infected, are treated surgically. The other lesions need only antiseptic precautions to prevent infection. Among these, general cleanliness is the most important, and, perhaps chiefly for that effect, sea-bathing is highly recommended.

XVI.—SYPHILIS

Synonyms: *Lues, Lues veneris.*

Syphilis (Gr., *συς*: swine; *φίλος*: a swine lover, a term coined by the poet, Fracastoro, in 1530) is a specific, inoculable, constitutional disease which in its earlier course presents symptoms of systemic inoxication analogous to the symptoms of acute infectious fevers, especially the exanthemata, and which in its later course presents circumcribed lesions having the characteristics of infective granulomata, like uberculosis and leprosy.

There are references to a disease which can be identified with syphis in early Chinese and Indian literature. In spite of the fact, howver, that it is a disease of the greatest public importance, and one of hich mention ought to be found in the literature of any country where exists, it is impossible to identify syphilis with any disease mentioned n mediæval or ancient European literature. The disease appears upon he stage of history with dramatic suddenness in a malignant epidemic hich scourged Europe during the last years of the fifteenth century, nd which can be traced to the French and Spanish campaigns in orthern Italy in 1494–95. Its sudden appearance in history at this ime, just after the first voyagers to America had returned to Europe, as led to the belief that the disease was brought to Europe from Amerca by the sailors of Columbus, but demonstration of the fact is lacking, s is demonstration of prehistoric American syphilis.

Syphilis and the other venereal diseases were for a long time inexricably confused. The first great fact to be established in the delimitaion of syphilis was the determination by Ricord, the great French yphilographer, that syphilis and gonorrhea were separate diseases. Then nother Frenchman, Bassereau, established, in 1852, the distinction beween the hard or syphilitic chancre and the soft chancre, and with he establishment of that fact the conception of syphilis became clear.

THE COURSE

The course of syphilis falls naturally into several stages:

(1) A first period of incubation, from the time of infection to the ppearance of the initial lesion.

(2) A second period of incubation, or *a primary stage*, from the ppearance of the initial lesion to the development of cutaneous lesions.

(3) *A secondary stage*, from the appearance of cutaneous lesions ntil the disappearance of evidences of an active constitutional infecious disease.

(4) A so-called *tertiary stage*, after the disappearance of evidences f general infection, which may or may not occur and which is characerized by circumscribed gummatous lesions.

The first and second periods of incubation and the early secondary tage are definite and fixed and invariably occur in regular sequence. he tertiary stage is not so sharply defined. It may be better regarded s the stage of gummata or of the formation of granulomatous deposits;

and while these lesions, as a rule, occur after the secondary stage, they may occur precociously along with secondary lesions. The tertiary stage, therefore, does not always occur in definite sequence, and is not always sharply separated from the secondary stage.

First Incubation Period.—From the date of inoculation to the appearance of the initial lesion is, in the vast majority of cases, from twelve to twenty-one days, usually about two and a half to three weeks, but the extreme limits of the primary incubation period are from ten to seventy days (Taylor). Great variations from the average incubation period are, however, excessively rare, and most of them are open to suspicion.

The second incubation period, or the primary stage, lasts from the appearance of the chancre until the first appearance of cutaneous lesions. It has been observed to be as short as twelve days (Gibert). It is usually forty to forty-five days, about six weeks; sometimes sixty, and very rarely ninety days. It is apt to be prolonged by hot weather and shortened by cold, and it may be prolonged in cachectic subjects. As a rule, the initial lesion persists through this stage.

In the beginning of this stage there are no constitutional symptoms; nothing but an indolent, painless, and usually trivial-looking ulcer. Gradually, in the course of ten days or more, lymphatic engorgement appears in the contiguous glands. Before the appearance of cutaneous lesions the lymphatic engorgement becomes general, with enlarged, painless glands. At the same time evidences of systemic intoxication begin to appear. These may be trivial or absent, but patients are usually conscious of some indisposition.

Secondary Stage.—The secondary stage is fully established with the appearance of the eruption, and the limits of its duration are from a few weeks to a year or more, usually several months. The demonstrable evidences of the existence of this stage subside gradually, so that its end is not sharply defined.

During the course of secondary syphilis patients show to a greater or less extent the symptoms of a constitutional infectious disease. In addition to the lesions of the skin and mucous membranes and the general adenopathy there are more or less general depression or cachexia, fever, rheumatoid pains, insomnia, anæmia, and other evidences of toxæmia. These constitutional symptoms are usually slight, and frequently, aside from some feeling of general depression, are entirely absent. In a certain proportion of cases, however, they are very distinct, and in rare cases their manifestations are intense. The general depression of syphilis, or the syphilitic cachexia, consists of such symptoms as accompany toxic fevers and anæmia: a certain amount of loss of appetite, weakness, dejection, nervousness, and perhaps emaciation and pallor.

These symptoms may occur in the beginning of syphilis or may develop several months later. They are seen especially in persons who are not vigorous and in those who are poorly treated.

During the most active period of secondary syphilis there is, as a rule, a specific fever, but it may not occur. If present it is usually mild, showing an evening rise of one or two degrees; but it may be well marked with a morning temperature of 102° F. and an evening temperature up to 104° and 105° F., in extreme forms simulating closely malaria, typhoid fever, or tuberculosis. It appears occasionally before the occurrence of the eruption, but most frequently it is not noticed until the eruption has developed. It may be continuous, remittent or intermittent throughout the secondary stage, or it may occur at any time during the secondary stage.

There is usually anæmia, with more or less pallor. The cellular elements of the blood do not show special changes, but the number of red cells is reduced perhaps to three millions or less, the hæmoglobin is diminished, and moderate leucocytosis occurs. The anæmia persists during the active period of constitutional syphilis, and disappears as the active evidences of the disease subside. When mercury is administered in the active stage of secondary syphilis there is at first a sudden decrease of the hæmoglobin—ten to thirty per cent, Gower's scale—but this disappears in a few days, and under continuation of mercurial treatment the hæmoglobin increases and the diminution becomes gradually less marked as the disease vanishes (Justus's test). Justus's test was suggested as a diagnostic measure, but it has not proved reliable. Winternitz has found an increase in fibrinogen in the blood of recent syphilis.

Acute nephritis may occur in the secondary stage, usually within the first six months, but occasionally later. According to Lafleur, it occurs in about 3.8 per cent of cases. It may persist for several months, but it usually disappears, and is very rarely followed by chronic nephritis. In a few instances it has been fatal in two or three weeks.

Hæmatogenous jaundice of slight degree is not infrequent, occurring at any time during the secondary stage, unassociated with demonstrable hepatic changes, and lasting from a few weeks to two or three months. Occasionally it is intense. In very rare instances acute yellow atrophy of the liver occurs.

Other very rare complications are pleurisy and angina pectoris, epididymitis, and parotitis.

Pains in the muscles, bones, fascia, and joints, most pronounced in the extremities, are among the frequent symptoms in the early months of secondary syphilis. The sensation varies from a feeling of weakness or slight soreness to dull or severe aching pains. The pains usually come on toward evening, are worse at night, and disappear toward morning, leaving a feeling of stiffness or soreness. A nocturnal headache is also a frequent symptom. It is usually mild, but is occasionally distressing. It may be temporal, frontal, or occipital, and is occasionally a sharp, localized pain. Both the headache and the rheumatoid pains usually develop in the early secondary period, but may recur later. Neuralgic pains, particularly in the fifth cranial nerves, sometimes occur, especially in patients who have previously suffered from neuralgias. A certain amount of insomnia is not uncommon, independent of any pain to account for it. It is peculiar in that it is not readily influenced by soporifics, but ceases under mercurial treatment.

Of the organs of special sense, the eye is the most frequently involved in secondary syphilis. The usual lesion is an iritis, which as rule invades one eye, and then the other, and appears in the third t seventh month of the disease. It may be very mild, with slight ciliar congestion, or extreme, causing great pain. Choroiditis and retiniti are rare secondary symptoms. Very rarely papules appear upon th conjunctivæ, and, in the rarest cases, even upon the corneæ. The middl ear is, in extremely rare cases, affected in secondary syphilis by extensio of the disease from the nasopharynx.

It should be emphasized that, as a rule, the constitutional symptom of secondary syphilis are mild, and in themselves trivial. Apparentl severe constitutional symptoms were much more frequent formerly tha they are at the present time. We seldom meet with cases in which th constitutional symptoms are troublesome except in old alcoholics and i individuals poorly nourished, untreated, and living under bad hygieni surroundings; and when one does meet such cases, the symptoms, as rule, yield promptly under appropriate treatment. In very rare cases however, the constitutional symptoms of secondary syphilis are greatl intensified; with or without exaggeration of the skin and mucous membrane lesions, constitutional symptoms of marked severity arise, and i rare instances produce fatal results. Such cases may occur in thos who are robust and vigorous, and have their origin in infection fro cases of ordinary type. The severity of these cases is apparently accounted for by individual idiosyncrasy, which in some instances amount to almost complete inability to resist the infection. The severe symptoms usually manifest themselves early in the secondary period. It i to cases of this type that the term *malignant syphilis* is applied, an not to the exaggerated cases of destructive tertiary lesions which ar sometimes seen as a result of lack of treatment.

Tertiary Stage. The tertiary stage of syphilis is the stage of gummatous lesions, and is hardly a chronological stage. Its lesions ma occur during the period of active secondary syphilis. As a rule, the

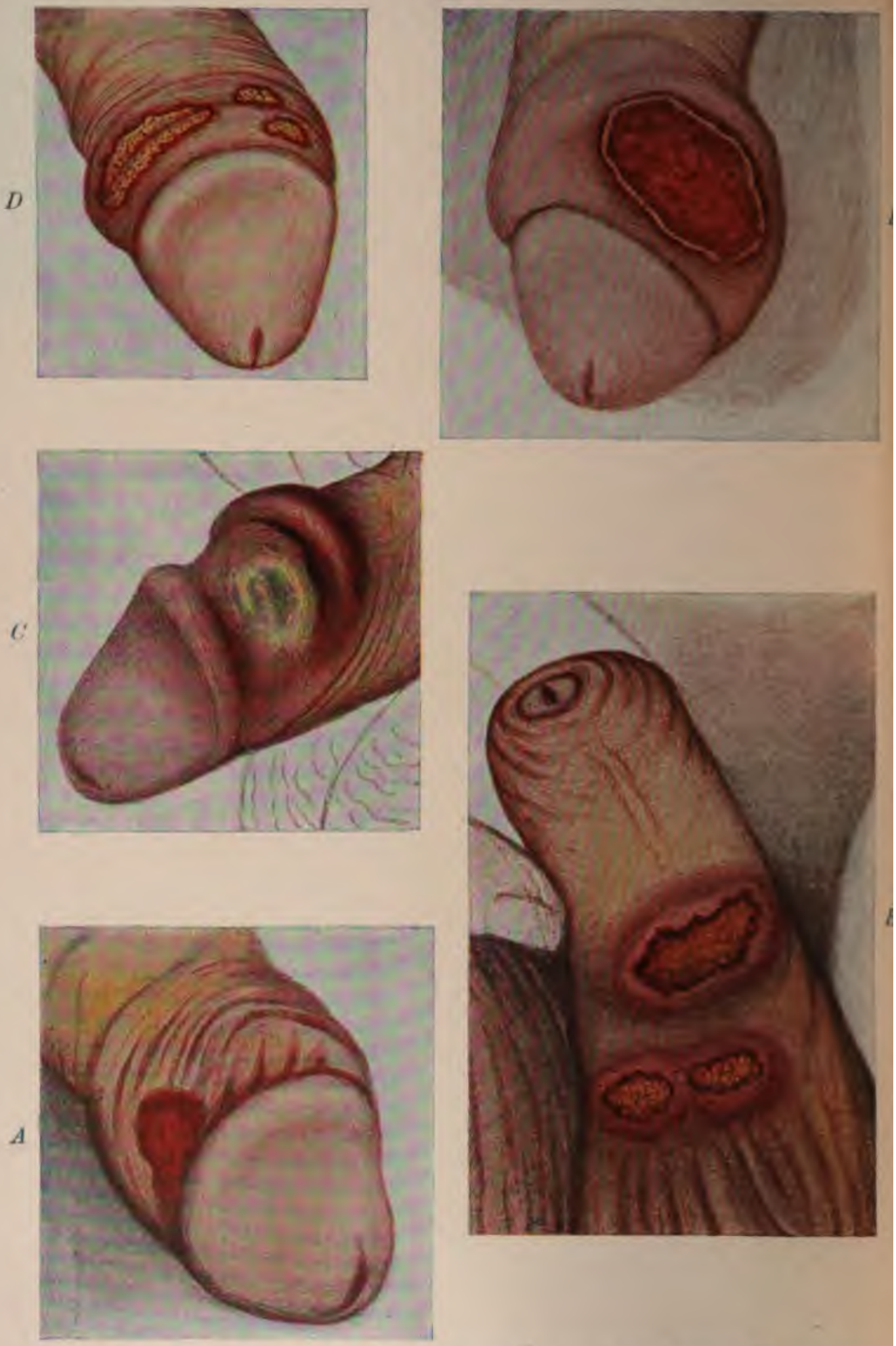

FIG. *A*, CHANCROUS EROSION. FIG. *B*, EXCORIATED CHANCRE WITH DIFFUSE INFILTRATION. FIG. *C*, HARD CHANCRE IN PROCESS OF REPAIR. FIG. *D*, YOUNG CHANCROIDS. FIG. *E*, CHANCROIDS.

These illustrations are from unpublished water-colored drawings of Dr. R. W. Taylor's, which *with great* generosity he has placed at my disposal.

occur after the disappearance of active evidence of constitutional syphilis, and there is a possibility of their occurrence at any subsequent time during the life of the patient. The lesions of tertiary syphilis are granulomata, which occur as circumscribed neoplasms and produce the symptoms of tumors. They may occur in any of the tissues, and the symptoms which they cause depend upon their location. In the skin they can be diagnosticated by their peculiar characteristics, but occurring in the deeper tissues they may present no features to differentiate them from other tumors except their amenability to antisyphilitic treatment.

Quaternary Stage.—Finally, reference is sometimes made to a quaternary or parasyphilitic stage in which, years after the disappearance of active syphilis, certain diseases may occur that, as experience shows, have a definite relation to preceding syphilis. The chief general affections of this sort are locomotor ataxia and paralytic dementia; a local parasyphilitic lesion is leukoplakia which occurs on the oral mucous membrane. What the relationship is of these parasyphilitic diseases to syphilis is not known. They are not a part of the disease, and are not influenced by antisyphilitic treatment, a characteristic which is strongly marked for all of the lesions of the three stages of actual syphilis.

THE CHANCRE

SYNONYMS: *Initial Lesion, Primary Lesion, Hard, Hunterian, or Infectious Chancre, Initial Sclerosis.*

The first evidence of the development of syphilis is the chancre or initial lesion. It may be said that syphilis practically always begins with a chancre, although the possibility of direct blood inoculation without initial lesion must be admitted. Thus Jullien [1] records the case of a surgeon who became infected by direct blood inoculation in an operation and, without having a primary sore, developed secondary syphilis within thirty days.

The Initial Lesion.—The initial lesion of syphilis begins, usually two to four weeks after infection, as a red spot or papule which develops in the course of a few days into a hard infiltration in the skin. This infiltration may be either a papule or a flattened plaque. Usually it is a round or flattened papule from the size of a small pea to that of a bean. It may either be imbedded in the skin without elevation, or it may project above the surface in the form of a well-defined swelling. In either case it is a sharply defined neoplastic infiltration which can readily be felt between the fingers. It is of red or dull red color, indolent, painless, and hard. Its later appearance depends upon circumstances.

Upon surfaces where the lesion remains dry it may never become eroded, but persists throughout as a dry papule or plaque covered with scaly but not broken epidermis. This is the so-called *dry, indurated*, or *scaling papule*, an uncommon form of chancre.

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[1] *Brit. Jour. Derm.*, vol. xiii, 1901, p. 398 (abstract).

In a large proportion of the cases the epidermis is destroyed over the top of the papule, leaving the central part of the lesion eroded, and forming the so-called *superficial* or *chancrous erosion*. This lesion and the Hunterian chancre—yet to be described—are the typical chancres. The chancrous erosion has a base of red or dull-red color which may amount to a well-developed globular papule or nodule, or may be a large, flattened, disk-shaped papule, or a thin, parchmentlike superficial layer of induration in the corium with little or no elevation. The central part only of the lesion is eroded, forming a superficial ulceration from an eighth to half an inch or more in diameter, round or oval, perhaps slightly excavated, or, in some cases, even projecting above the surrounding rim of the lesion. The eroded surface is smooth and glistening, without granulations, and with a scant seropurulent exudate.

In the third form of initial lesion—the *Hunterian* or ulcerated chancre—there is, instead of the central erosion, central ulceration, the whole lesion resembling the eroded chancre, except for the fact that there is upon the top of the plaque or tubercle a more or less deep saucer-shaped ulcer with sloping margins. The ulcerated surface is smooth and covered with a necrotic membrane and there is a scant seropurulent exudate.

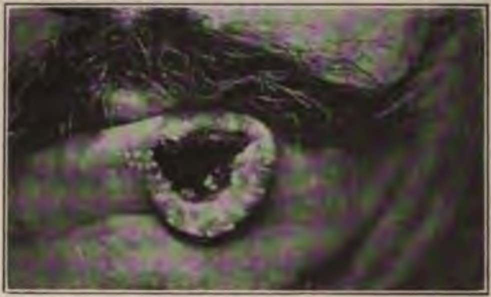

FIG. 134.—CHANCRE OF LIP. (Grover W. Wende's collection.)

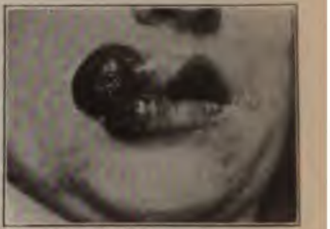

FIG. 135.—THREE CHANCRES OF LIP. (Howe's collection.)

In all of these forms of chancre the most characteristic objective feature is the indurated base; the excoriation or ulceration is secondary. This induration is due to a dense, round-cell infiltration of the corium, especially around the blood vessels. If the infiltration is very superficial the induration is spread out as a thin sheet, like a disk of writing paper (laminate induration), or, if a little thicker, like a piece of parchment (parchment induration). Usually it produces either a disk-shaped or a nodular tumor which feels like a neoplasm in the skin, is sharply circumscribed, and is densely hard—as hard as cartilage or as a piece of hard paraffin. Under pressure the blood is readily expressed from the indurated tumor, and the lesion stands out like an ivory-white circumscribed mass in the skin. The induration reaches its maximum about two weeks after the appearance of the lesion.

The next most characteristic feature of the chancre is its indolent course. It is associated with almost no discomfort and persists with little tendency to heal, if treatment is not given, for from four to six weeks, frequently until the appearance of the eruption. After the healing of the chancre the induration usually persists for several months; it may even persist for years or become permanent as a circumscribed sclerotic mass in the skin.

Mixed Chancre.—At the time of inoculation with syphilis there may be inoculation with common pus organisms or with the organism of chancroid. It that event there may develop within two or three days an infected sore which will pursue the ordinary course of a simple infection or of a chancroid. In such a case the chancre develops at its usual time. Its appearance may be entirely masked by the destructive ulceration of the other infection, but if this subsides, as it may, in the mean time, the lesion usually takes on the normal appearance of the Hunterian or ulcerating chancre. The mixed chancre is a source of much confusion in diagnosis. It makes it impossible to say that what is apparently a chancroid will not be followed by syphilis, because one can never know that infection with syphilis has not occurred at the time of infection with chancroid.

In a large majority of cases the initial lesion of syphilis is single. If the inoculation, however, occurs at multiple points, two or more chancres may develop. If so, they develop at the same time, never in sequence, and they are all of equal age.

The usual location of chancres is about the genitals, but they have been seen in almost all conceivable locations. After the genitals they are most frequent upon the lips; next, perhaps, in the mouth or upon the hands (especially of doctors and dentists). Extragenital chancres, particularly those about the lips and mouth, are usually larger, ulcerate more deeply, and are altogether more angry-looking lesions than the typical genital chancre, but except for these exaggerated features they present the usual characteristics of the lesion. There is a common impression that syphilis following extragenital chancre is apt to be more severe than after genital chancres. There seems no good reason why this should be so, and my experience does not confirm it.

The most important diagnostic features of a chancre are its long period of incubation, its persistence with comparatively little change for two or three weeks, its noninflammatory indolent character, the superficial trivial ulceration, the induration of the base, and the absence of subjective symptoms. Of these the most definite and valuable are the long period of incubation and the long persistence of the lesion. All of the other characteristics are liable to greater or less variation which may obscure them. Any indolent lesion with superficial ulceration and with little inflammatory reaction which appears about the genitals, or indeed elsewhere, and persists almost unchanged for several weeks, should make one suspicious of chancre. As a matter of fact, the characteristics of an unmixed chancre are so definite in most cases that an expert can with almost complete assurance determine its nature. On the other hand, in atypical cases this is not possible, and, because of the possibility of error, one should never make a positive diagnosis upon the chancre alone. A probable diagnosis can be made in most cases, but the demonstration of the syphilitic character of the lesion is not complete until the eruption appears.

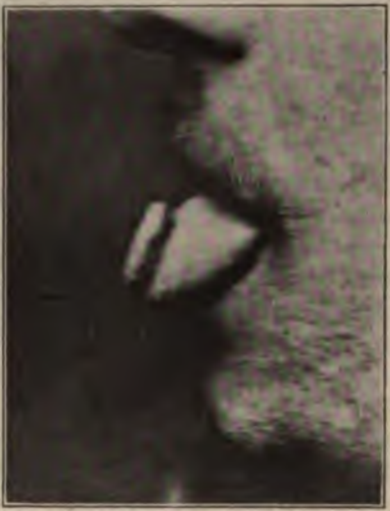
Fig. 136.—Chancre of Tip of Tongue.
(C. J. White's collection.)

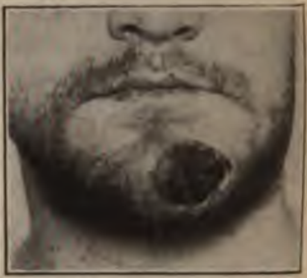
Fig. 137.—Chancre of Chin.
(Whitehouse's collection.)

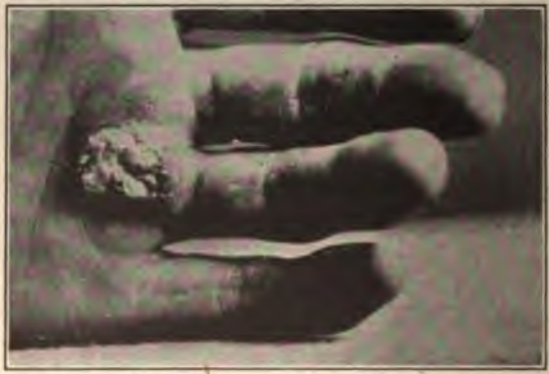
Fig. 138.—Chancre of Palm in a Dentist.
(Grover W. Wende's collection.)

The lesions which are likely to be confused with chancre about the genitals are herpes, and chancroid, and occasionally epithelioma. Chancroid lacks a definite period of incubation, and usually appears in two or three days; begins as a pustule and develops into a round or irregular excavated ulcer with abundant purulent autoinoculable secretion. There is inflammatory induration, but not the dense, hard induration of chancre. The ulcers are often multiple, they are more or less painful, and pursue an irregular course.

Herpes about the genitals begins, a day or two after irritation, as vesicles which quickly rupture and leave superficial, round, painful excoriations. These are grouped together and form small, polycyclic, abraded patches. Their surface is bright red, or sometimes covered with a necrotic pellicle, and, unless they become infected, they heal quickly.

Extragenital chancres are usually as easily diagnosticated as those in typical locations; occasionally on the lips the diagnosis has to be made from epithelioma. The difference in the characteristic borders of the two conditions usually leaves no difficulty in diagnosis, but in doubtful cases the positive diagnosis depends upon microscopical examination of tissue to determine epithelioma, or upon the occurrence of secondaries to establish syphilis. Lesions in the mouth and nose sometimes may be confused with diphtheria. The indurated, elevated base is usually sufficient to make the diagnosis of syphilis clear, but in rare instances culture experiments are necessary to exclude diphtheria.

Adenopathy.[1]—More or less general enlargement of the lymphatic glands is a characteristic feature of syphilis and is, after the chancre, the first objective evidence of syphilis. The glands adjacent to the chancre are involved first and become palpable usually from the seventh to the tenth day, but at times as early as the fifth or as late as the fourteenth day. They are usually multiple and bilateral, but they may be unilateral or single. They are palpable as oval or rounded, painless, firm, subcutaneous nodules, from the size of a pea to that of a lima bean or larger. At the same time there is frequently enlargement of the perivascular lymph spaces adjacent to the initial lesion, so that the lymphatics may be felt as a cord or cords leading from the chancre for two or three inches, but seldom as far as the adjacent ganglia. Two or three weeks after the enlargement of the glands adjacent to the chancre—that is, from one to three weeks before the appearance of the eruption—general enlargement of the lymphatic glands occurs, not only of the subcutaneous glands but of the visceral glands as well. The glands most frequently appreciably affected are those of the posterior and anterior triangles of the neck, the epitrochlear, and the axillary; the most characteristic of these are the posterior cervical and the epitrochlear. Frequently several glands are to be felt on either side of the neck and in the axillæ. Usually there is only one epitrochlear palpable behind either elbow, but occasionally two or three may be felt. In addition there may be palpable enlargement of the glands over the mastoid process, and of the submaxillary, submental, occipital, femoral, and intercostal glands.

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[1] Berg, *Jour. A. M. A.*, July 14, 1906, p. 156 (abstract).

The glandular enlargement is characterized by induration, persistence, and absence of inflammation. The enlarged glands are usually small, from the size of a pea to a hazelnut; occasionally they are as large as a pigeon's egg or even larger. They are painless, not tender, and indolent. They remain enlarged during the eruptive stage and very slowly disappear. Like other evidences of syphilis their disappearance is greatly hastened by the administration of mercury. Exceptionally certain glands, especially the inguinal and posterior cervical, remain enlarged for years.

The diagnostic importance of general adenopathy in syphilis is great if the lymphatic engorgement has developed in regular sequence after a suspicious sore. In individuals who have not a superfluous amount of flesh the enlarged glands can practically always be found in early secondary syphilis. Failure to detect them, however, does not exclude syphilis in fleshy persons in whom their palpation may be difficult or impossible. On the other hand, the discovery of enlarged lymphatic glands is not of great significance as an evidence of syphilis unless they occur in proper association with the other cardinal manifestations of secondary syphilis; for, especially in thin persons, it is usual to be able to palpate some of the glands that are sought for in syphilis. Dietrich found in 439 cases of healthy individuals palpable glands somewhere in ninety-nine per cent, palpable cervical and supraclavicular in seventy-nine per cent, axillary in seventy-two per cent, epitrochlear in eighty-two per cent, and inguinal in ninety-three per cent.

Cutaneous Lesions of Syphilis

Synonyms: *Syphilides, Syphilodermata.*

Secondary Lesions.—Syphilis produces in its secondary stage cutaneous lesions of all morphological types from macules to nodules. These different forms of lesions may, and frequently do, occur in combination, so that the eruptions of syphilis present many different clinical pictures. In all of them, however, are found certain common characteristic features.

Characteristics.—The macular eruption of syphilis is a congestive erythema of toxic origin, similar in character to the other toxic erythemas. All of the other cutaneous lesions of syphilis, from papules to nodules or gummata, are of essentially one character. They are sharply defined, round-cell infiltrations (granulomata) in the papillæ and corium, and their various objective characteristics depend upon this fact. Kaposi has analyzed these characteristics as follows:

(1) The syphilides are sharply defined, dense, and uniform cellular infiltrations of the papillary body and corium, and differ from one another only in size.

(2) The cells of which they are composed are not fitted to undergo permanent organization into connective tissue, but always undergo involution and disappear, either by absorption or by purulent degeneration.

(3) The syphilitic infiltration of the skin always enlarges and disappears centrifugally. Hence the peripheral parts are relatively the most recent and exhibit the characteristics of the fresh infiltrations, while the oldest parts are in the center and are the first to disappear.

From these essentials the characteristic features of syphilitic lesions can be made out. They can best be illustrated, as Kaposi illustrated them, by a consideration of the syphilitic papule, which may be regarded as the typical syphilide.

As a result of the dense, sharply defined infiltration the papule projects. The epidermis over it is stretched and glistens. It is firm to the touch and, while under pressure the redness disappears, the evidence of infiltration remains. The color is brownish red, on account of the escape of blood pigment due to stasis in the compressed vessels of the lesion. After a longer or shorter interval retrograde changes and absorption occur, resulting in a depression almost microscopic upon the top of the papule. The epidermis wrinkles at this point and breaks down into scales, while the periphery of the papule retains the characteristic features of the infiltration—that is, it remains brownish red, firm, tense, and glistening. Instead of undergoing fatty degeneration and absorption, necrosis and ulceration may occur. Then a central ulceration takes the place of the scaling, atrophic center. There is first, perhaps, a central pustule on the tip of the papule which subsides into a crust, corresponding to the central oldest part of the infiltration, and is surrounded by a border of brownish-red infiltration.

The same changes that are seen in miniature in the papule occur in the nodular infiltrations or gummata. When the gumma breaks down it breaks down at the center with the formation of a central ulcer surrounded by a border of dense, smooth, brownish-red induration. If there is a single nodule a round ulcer is formed. If the nodules are multiple and confluent there is formed an ulcer of polycyclic or circinate outline upon the elevated indurated base. But in all cases there occur the fundamental characteristics of central involution or ulceration with the spread of the infiltration, if it spreads, at the periphery.

In addition to the fundamental features of Kaposi the syphilides have certain other characteristic features.

Arrangement.—The lesions tend to occur in groups and to be arranged in circles or circinate figures. This tendency is slight in the earliest lesions, but becomes more and more marked with the age of the disease.

Distribution.—The eruptions of the secondary stage, like all eruptions due to general infection, tend to be generalized, bilateral, and symmetrical. The tertiary eruptions, on the other hand, are usually localized, of limited extent, and asymmetrical. In distribution the secondary syphilides show a well-marked predilection for certain parts: the face, especially on the forehead and around the mouth and nostrils, the back of the neck, the front and to a less extent the back of the trunk, the flexures of the extremities, and the palms and soles. In these locations the eruption is usually well marked and most abundant, but lesions are scattered more or less abundantly over the intervening parts, not confining themselves solely to the sites of predilection.

Multiformity.—The presence of lesions of different ages and of different types is a common feature of syphilitic eruptions. This multiformity of lesions is more striking in syphilis than in any other disease except eczema. It is due to the fact that the eruption does not appear all at once and that lesions of one type develop into another, so that lesions in different stages of evolution and of different types occur together.

Color.—Upon first appearance syphilides are of bright or inflammatory red color; after a day or a few days their color becomes a dull or brownish red—the so-called copper color, or, better, raw-ham color, of syphilis. This color, however, is by no means characteristic, and is not entitled to as much consideration as is frequently given it. As the lesions disappear there is usually left a brownish pigmentation, due to the deposit of blood pigment in the tissues, which is slowly absorbed. This pigmentation also, while fairly characteristic, is not distinctive.

Subjective Symptoms.—Absence of subjective symptoms in the lesions is usually a characteristic of the syphilides. Ordinarily there is no itching, burning, or pain. Occasionally, however, in a florid eruption there may be some itching, and its presence should not be given too much weight as negative evidence. Relative absence of pain is also a characteristic of gummata and syphilitic ulcers, but here it is only relative and at times the pain may be considerable, especially in lesions on the legs.

The combination of all or most of these features in a given case forms a characteristic syndrome which is distinctive, and upon which more weight can be placed in diagnosis than upon any one or two of them alone. As in other diseases, one is much less liable to mistakes if he depends upon the entire symptom complex rather than upon one pathognomonic feature.

Varieties. MACULAR SYPHILIDE (*Erythematous syphilide, Syphilitic roseola*).—The macular syphilide is usually the first eruption of syphilis and is frequently the only eruption. It corresponds in character to the symptomatic toxic roseolas which arise in various infectious diseases or other forms of general intoxication. Pathologically it is a hyperæmia located in superficial areas in the skin, accompanied by the other evidences of a beginning inflammatory process. It is evident that it is a short step from this pathological process to the formation of dense, cellular infiltrations in the corium with the production of papules and the other morphological types of syphilitic lesions.

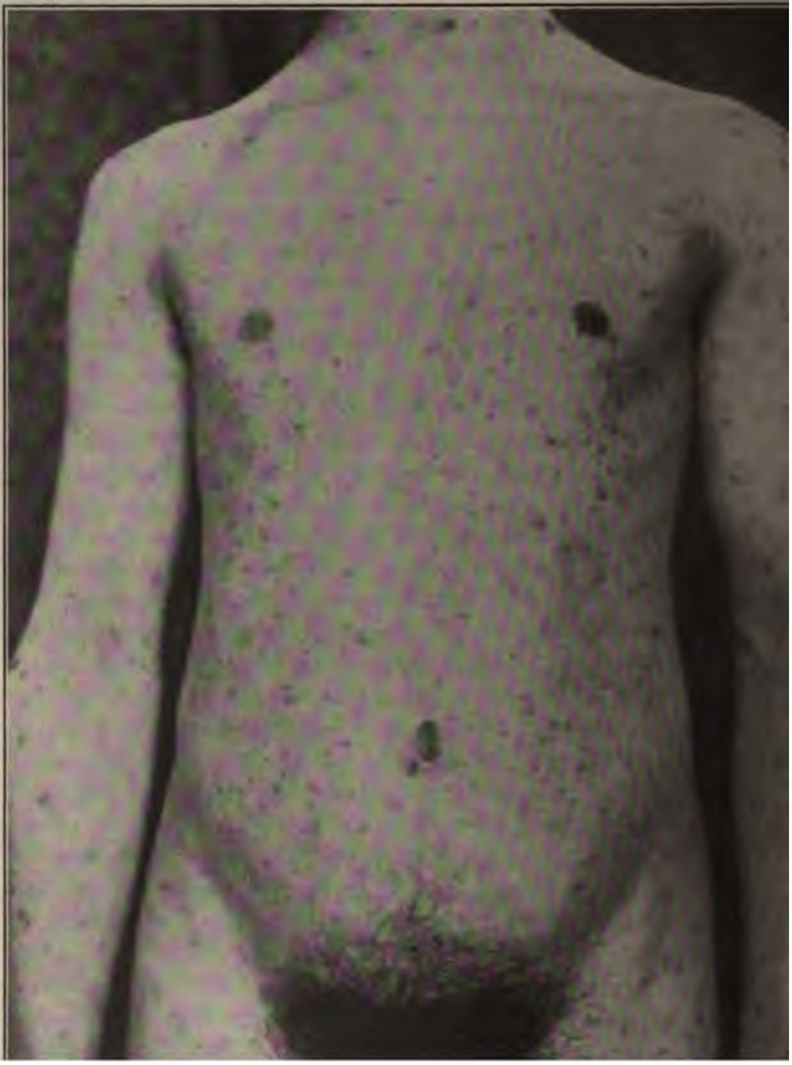
The macular syphilide appears usually from six to seven weeks, rarely as much as ten to twelve weeks, after the initial lesion. It appears as a diffuse mottling of the skin in ill-defined macules from an eighth to half an inch in diameter. They are at first a faint pink, but quickly become a duller red color, perhaps raw-ham color. Their color is of greatest intensity at the center and gradually fades off, so that their outline is ill defined. Under pressure the red color entirely disappears, leaving no trace of the redness, but leaving, perhaps, a yellowish stain due to blood pigment. The crop of macules is abundant, but their indistinct outline and faint color often make the entire eruption vague and indefinite in appearance, and on close inspection it may be difficult to distinguish the lesions from the normal mottling of the skin which occurs upon sudden exposure to the air. At a distance of four or five feet the eruption stands out more distinctly and is readily appreciable.

Frequently macules are the only type of lesion present. Some of the macules may have a small papule at the center, while in other cases many papular lesions will be intermingled with the macules, and in early eruptions in which papules or other infiltrated lesions are most abundant it is usual to find some macular lesions. In very rare cases well-marked œdema of the corium occurs in the macules, producing pink, elevated, œdematous lesions—the wheal type of syphilide.

The macular eruption is usually first seen on the front of the trunk, and it is characteristically most abundant on the front and back of the trunk and the upper two thirds of the extremities, especially on the flexor surfaces. Unlike the other secondary syphilides, it does not commonly involve the face, but if on the face it is most marked in the usual locations, on the forehead and around the mouth.

Syphilitic roseola usually appears quickly, frequently developing abundantly within twenty-four hours, but it ordinarily requires seven to ten days for its full efflorescence. It then persists for from one to

several weeks, or rarely for two or three months, and gradually d
pears. Occasionally it is followed by temporary pigmentation, but
not followed by desquamation. It may recur at any time withi



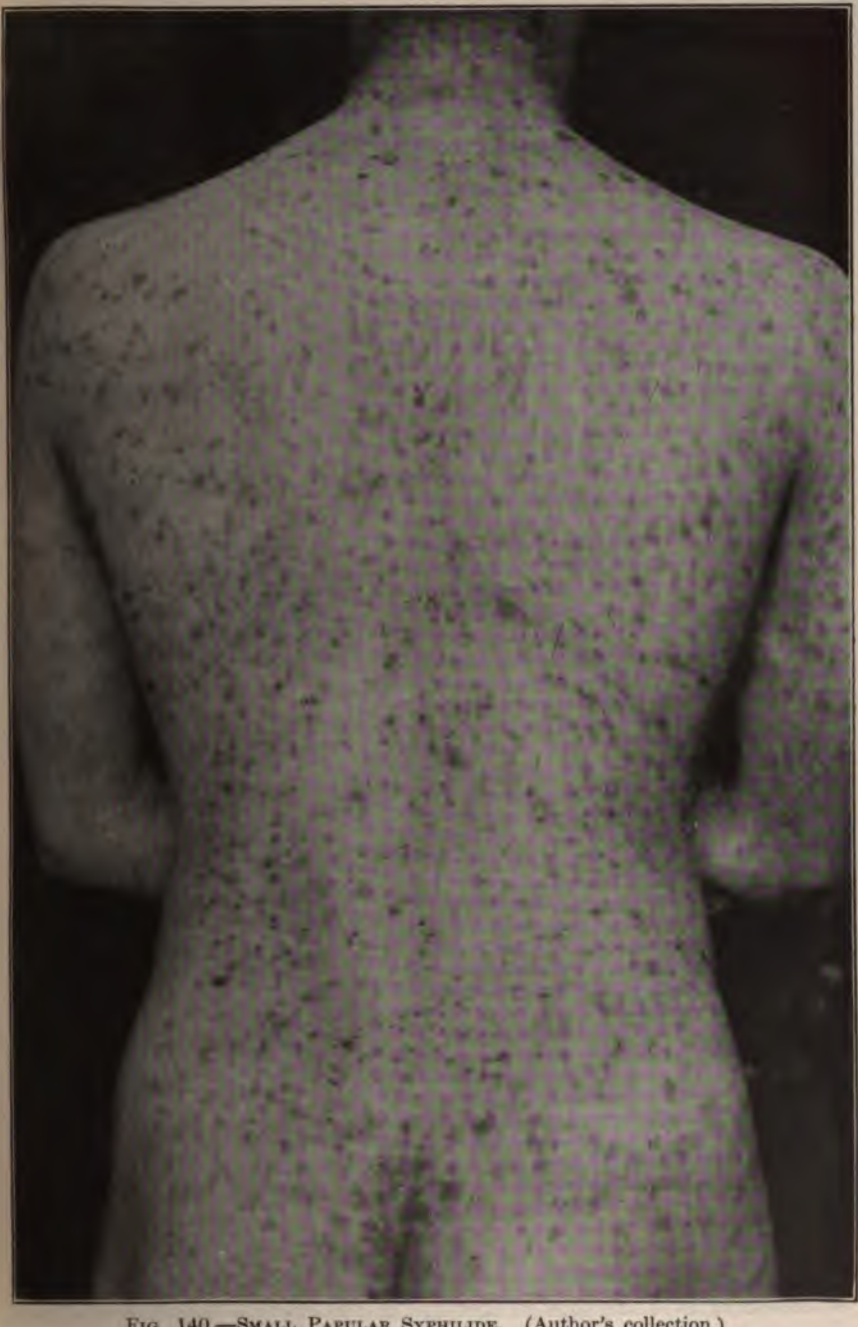


FIG. 140.—SMALL PAPULAR SYPHILIDE. (Author's collection.)

first year, but this is uncommon in treated cases. Rarely it recurs in the second and possibly in the third year.

Even when it occurs as an early secondary eruption some of the lesions frequently show a tendency to grouping in circinate figures, but ordinarily this tendency is lacking, and whatever arrangement the lesions show is in the lines of cleavage of the skin; in the line of direction of the ribs, for example, on the trunk. As a recurring eruption

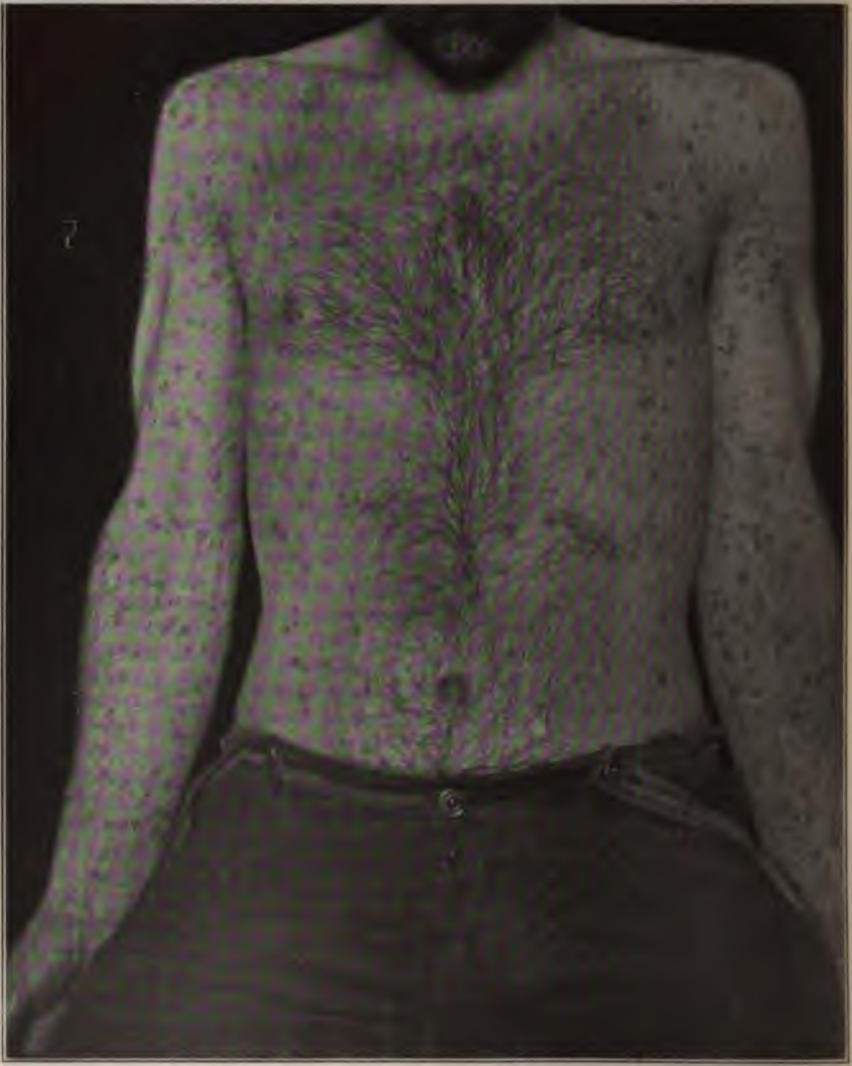


FIG. 141.—PAPULAR SYPHILIDE. (Author's collection.)

the lesions show a distinct tendency to group themselves in crescentic or circular figures.

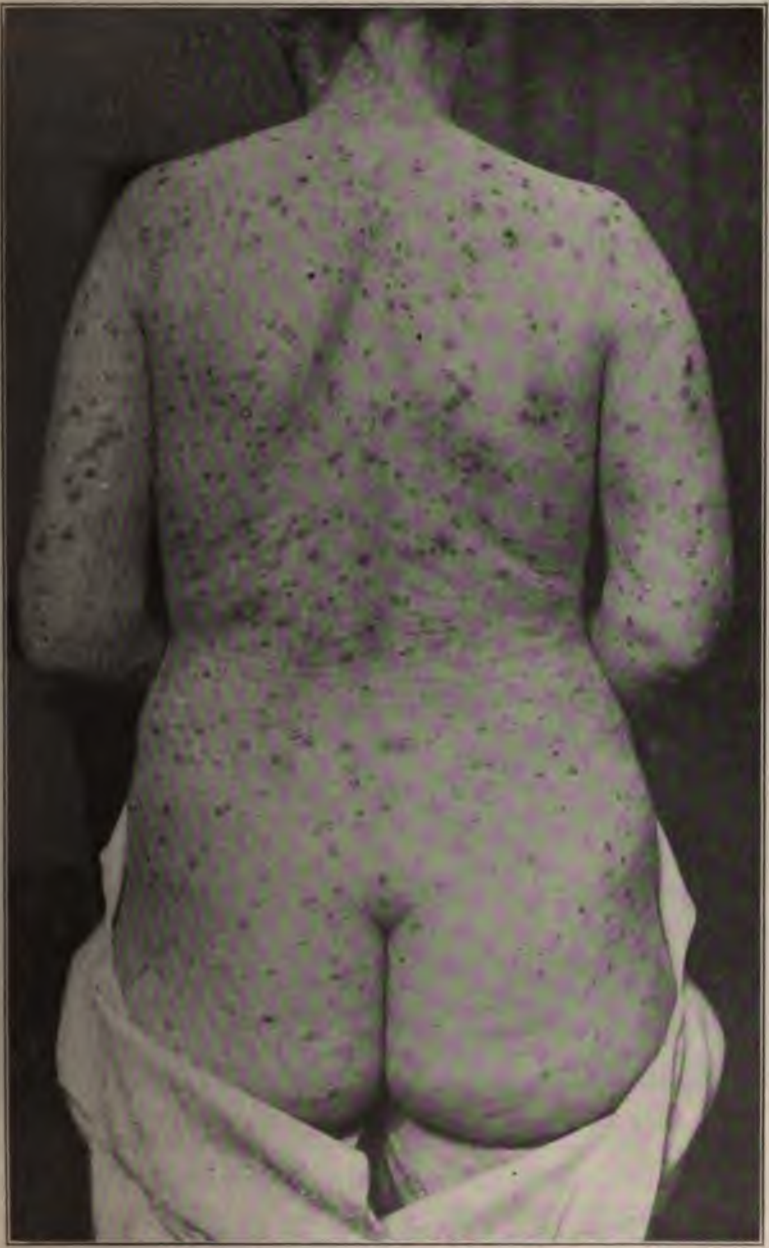


FIG. 142.—PAPULO-SQUAMOUS SYPHILIDE. (Author's collection.)

Syphilitic roseola is ordinarily unaccompanied by subjective symptoms, and frequently for that reason and on account of its inconspicuous appearance it is overlooked by the careless.

It is readily distinguished from the exanthemata and other forms of roseola by its association with other evidences of syphilis (especially recent chancre and mucous patches), by its slower development and longer persistence, by its greater abundance upon the covered parts, by the escape of the face and backs of the hands, by the absence of itching, and, upon its disappearance, by the frequent presence of stains and the absence of desquamation.

The uncomplicated macular eruption is usually the eruption of a mild form of syphilis. It is, in most cases which are promptly put under proper treatment, the only eruption. The papular and other eruptions characterized by dense cellular infiltrations are seen in association with syphilis of a severer type and most frequently in cases which are severer because of lack of treatment.

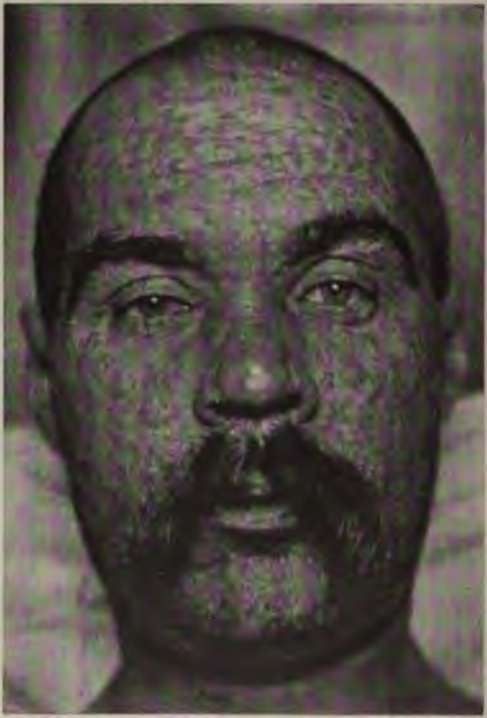


FIG. 143.—SMALL PAPULAR SYPHILIDE. Unusually abundant. (Author's collection.)

Papular Syphilides.—Papular syphilides are usually divided into several subvarieties according to their size, arrangement, and situation in the skin, but the differences between them are differences of minor detail, and all of them present the characteristics given in the above description of the typical papule. The chief distinction between papules depends upon their situation in the skin; the follicular or acuminate papular syphilide occurs around the follicles and is a conical papule; the flat papular syphilide occurs independently of the follicles as a rounded or flattened papule.

Follicular Syphilide (*Miliary papular syphilide, acuminate papular syphilide, lichen syphiliticus*).—The follicular syphilide occurs around the follicles, especially the hair follicles, and differs from the flat papular syphilide in being much smaller and in having a conical instead of a flat, rounded form. It is not a common form of eruption; is much less common than the flat papular syphilide, but is a very characteristic lesion. In size the papules vary from a pinhead to two or three times larger, and have the usual indolent red color of the other syphilides. They are frequently capped by a minute scale, and often

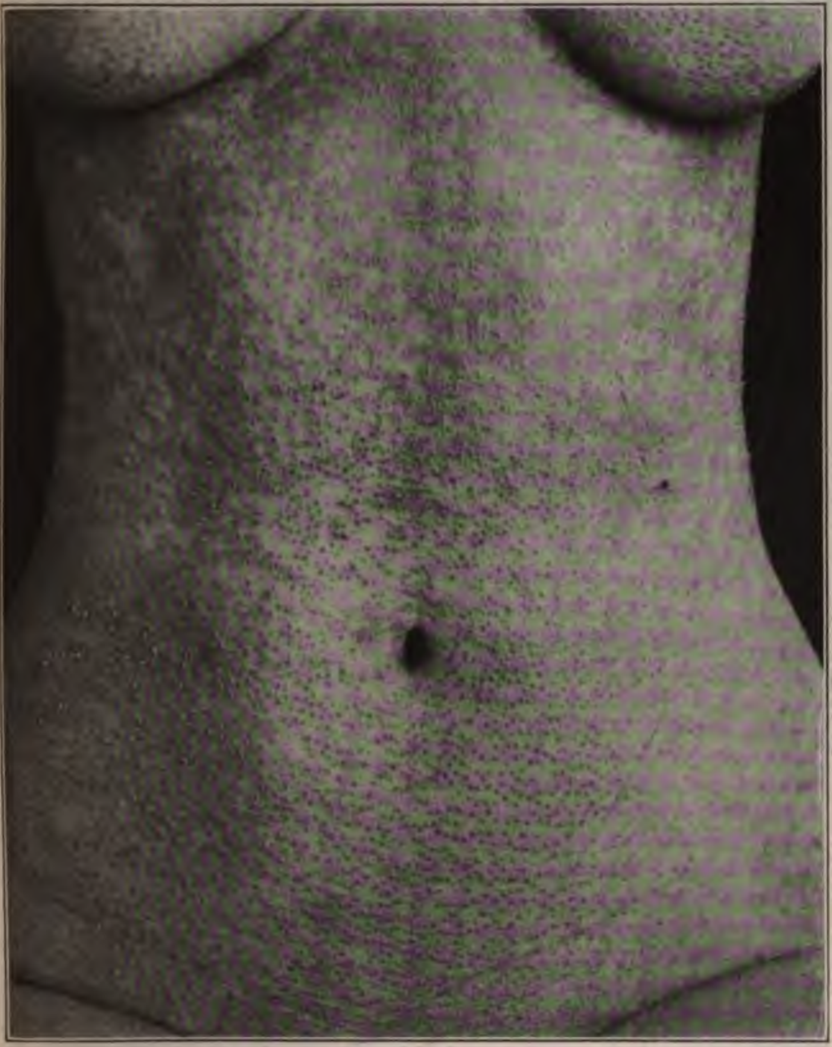


FIG. 144.—FOLLICULAR PAPULAR SYPHILIDE. (Howard Fox.)

upon their summits minute pustules form which quickly dry into crusts. They are usually associated with flat papules.

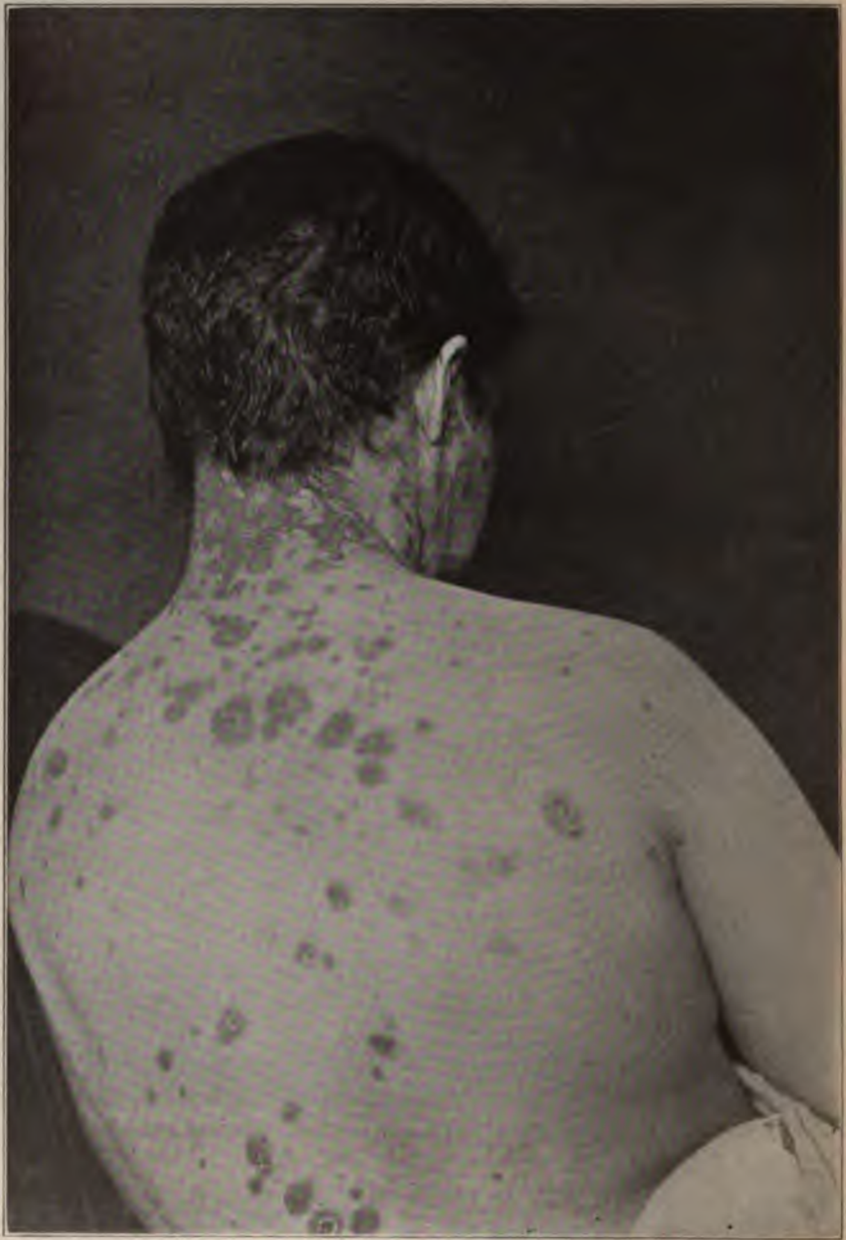


FIG. 144a.—LARGE PAPULAR SYPHILIDE, SHOWING CENTRAL INVOLUTION WITH TENDENCY TO RING FORMATION. (Author's collection.)

Their eruption is abundant and grouped, and the grouping has a tendency to circinate arrangement. The lesions are most abundant on the extensor surfaces of the arms and legs, on the back and sides of the trunk, and also at times on the face.

The follicular syphilide is usually an early secondary syphilide, appearing in the third or fourth month, but it may occur as a relapsing eruption any time during the active stage of the disease. It develops rapidly, becoming fully developed in the course of a few days to one or two weeks, and is very persistent, lasting from a few weeks to several months. Upon disappearing it leaves minute light-brown stains and at times atrophic pinpoint scars.

It is liable to confusion only with lichen scrofulosorum. From lichen scrofulosorum the follicular syphilide is distinguished by its association with other papular syphilitic lesions and with other accompanying evidences of acute syphilis, such as mucous patches and condylomata. Further, lichen scrofulosorum is a disease of childhood.

FLAT PAPULAR SYPHILIDE (*Lenticular syphilide*).—The flat, papular syphilide—the common type of papular syphilide—occurs in papules the size of a pinhead to that of a bean or larger, oval or round, flatly convex, abruptly but slightly elevated, firm and smooth, and of a deep-red or ham color. These papules are well-defined infiltrations imbedded in the skin. At first they are smooth and glistening, and they may never become scaly, but frequently individual lesions, or most of them after they have reached maturity, begin to desquamate, with the formation in some instances of thin exfoliating scales, in others of fairly abundant layers of scales upon the tops of the papules. The abundance of the eruption varies very greatly. At times it is not profuse although widely distributed, while in other cases it is very abundant. Even when abundant the lesions for the most part remain discrete.

Annular Syphilide.—The papules of syphilis show a more or less marked tendency to circinate grouping, and the groups may coalesce into crescentic or annular figures. In such a case one will see a partial or complete ring of the diameter of a coin or larger, made up of small papules, say an eighth of an inch in diameter, which are usually more or less scaly. This is one form of annular syphilide. Another form consists of a large, flat papule which spreads at the periphery while it subsides at the center. In this way there will be formed an annular lesion three eighths to an inch or more in diameter, which is made up of a rim of active infiltration with a subsiding center which is without elevation and shows more or less desquamation.

Corymbose Syphilide.—An occasional grouping of papular syphilides which is very characteristic is a large, bean-sized central papule surrounded by a group of smaller satellite papules—the corymbose syphilide.

The grouping may be distinctly circular with a central papule and a perfect encircling ring of satellite papules, or the satellite papules may be irregularly grouped around a larger central lesion. These lesions may be comparatively numerous. They are always associated with papular lesions not in corymbose groups.

The flat papular syphilide is usually most abundant upon the face, especially upon the forehead near the hairy border, forming the so-called *corona veneris*, and around the mouth and nose. Its other areas of predilection are the back of the neck, the back of the trunk, the flexor aspects of the limbs, the mammary folds, the groins, and around the genitals and anus. In these latter locations the lesions are usually converted into moist, vegetating papules, the so-called flat condylomata to be described later.

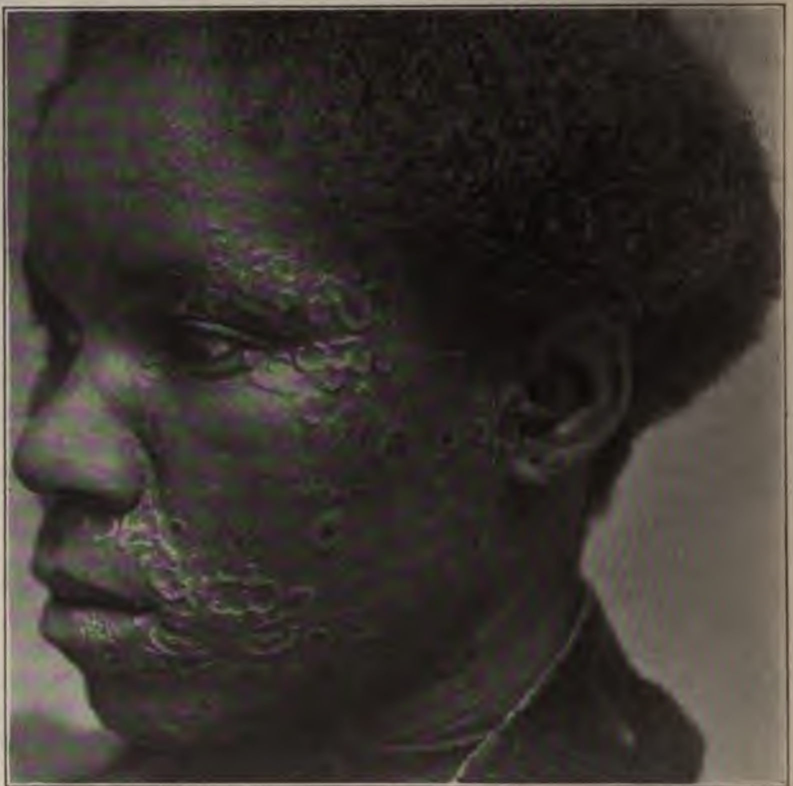


FIG. 145.—ANNULAR PAPULAR SYPHILIDE. This lesion is most frequent in negroes. (Schamberg's collection.)

A papular eruption may be the first eruption of syphilis, but it is more likely to follow a macular eruption, four or five months after infection, occasionally eight or even twelve months after infection. The eruption usually appears quickly, but requires from one or two to several weeks for its full development. Its course is rather indolent; it subsides under treatment in the course of a few weeks, but when left to itself may persist for months, and then is likely to be associated with ulcerative lesions. Upon subsiding it usually leaves pigmentation which is slow to disappear, and there may be atrophic scarring.

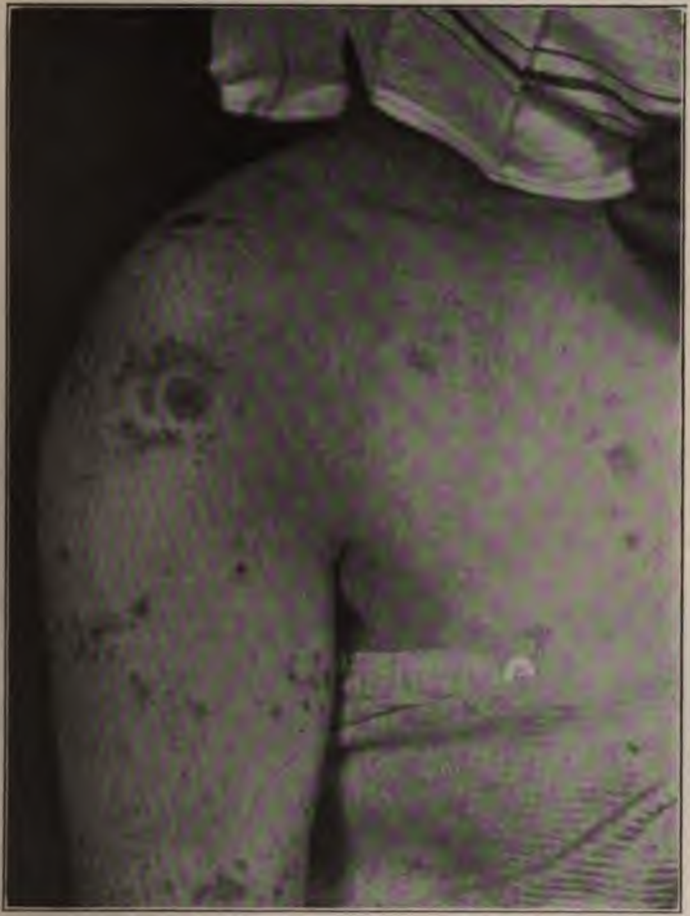


FIG. 146.—PAPULAR SYPHILIDE, SHOWING CORYMBOSE LESIONS, SIXTH MONTH OF THE DISEASE. (Author's collection.)

PALMAR SYPHILIDES.—In all of the secondary eruptions of syphilis palmar and plantar lesions are not uncommon, although they are by no means invariably present. On the palms and soles they are of the same character, and the description of the palmar lesions may suffice for both.

In these locations they usually take the form of deeply imbedded scaling papules. On account of the thickness of the horny epidermis the papules on the palms differ very much in appearance from the ordinary

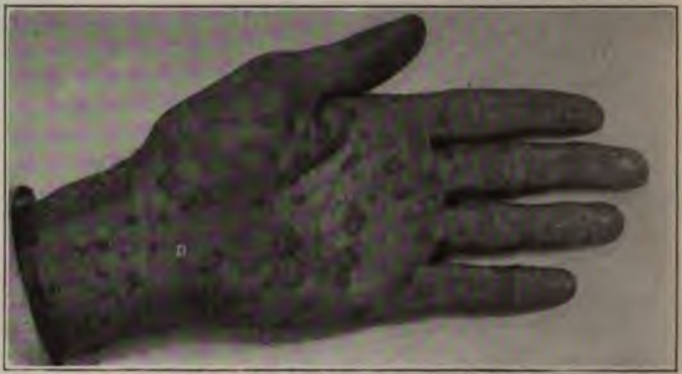


FIG. 147*a*.—SCALING PALMAR SYPHILIDE. (Author's collection.)

papular syphilides. The individual papules occur as deeply imbedded dark-red to ham-colored lesions, perhaps slight elevated, but usually depressed or at least without elevation. At first there may be no scaling but the epidermis over the papule quickly begins to exfoliate and form

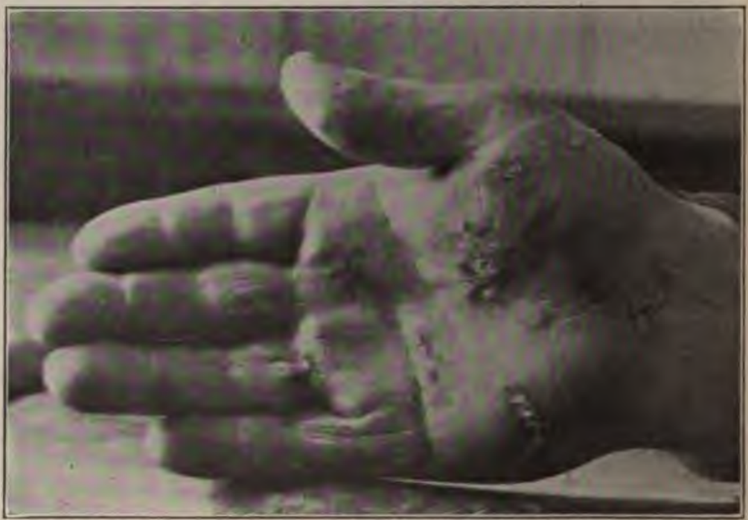


FIG. 147*b*.—PALMAR SYPHILIDE, DEPRESSED SCALING PAPULES. (Author's collection

a small circular area of scaling epidermis one sixteenth to one eighth of a inch in diameter, surrounded by a slightly undermined frayed collar o horny epidermis. In many cases the only palmar lesions are a few discret scaling papules such as these. Frequently the lesions are abundant upon the palmar surface, and then they are collected into circinate or round patches over which scaling occurs as it does in the individual lesions. The patches so formed are infiltrated and thickened, and may be covered with white, rather fine scales, or thin lamellæ. The borders of the lesions are sharply circumscribed, may show the peripheral irregularities of the coalescing papules, and usually have the frayed collar of uplifted horny epidermis. The scales upon the patches give them a yellowish-gray or whitish color, but upon removal of the scales the underlying patch is a characteristic indolent dull red. The infiltration of the lesions renders the skin inelastic, and as a result the formation of fissures is common.

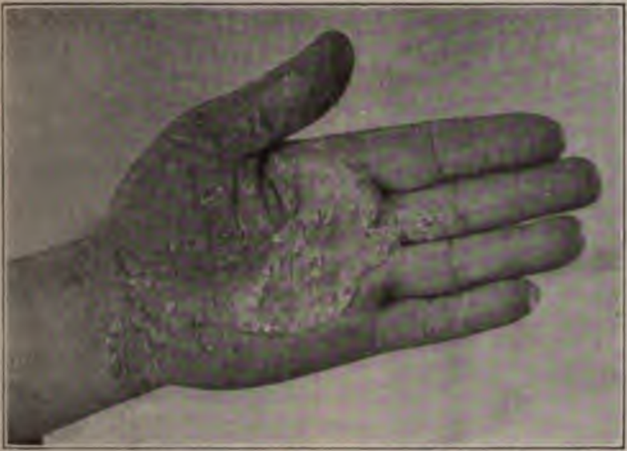


Fig. 147c.—Palmar Syphilide. Flat scaling papules, early secondary period. (Heidingsfeld's collection.)

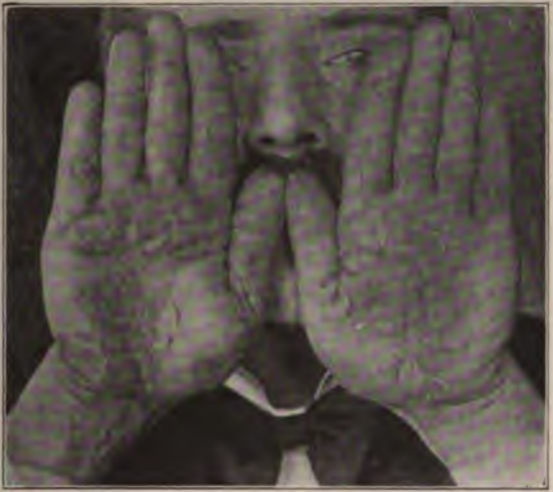


Fig. 147d.—Annular Papular Syphilide of Palms. (Whitehouse collection.)

The patches upon the palms may be single, but are usually multiple. Ordinarily they do not involve the entire palmar surface, but occasionally are so abundant that they become confluent and the entire palmar surface of the hand or of the hand and fingers is covered by one uniform, infiltrated, slightly scaling lesion. Without the fissures there may be no subjective symptoms or there may be slight itching. If fissures exist there is, of course, more or less tenderness.

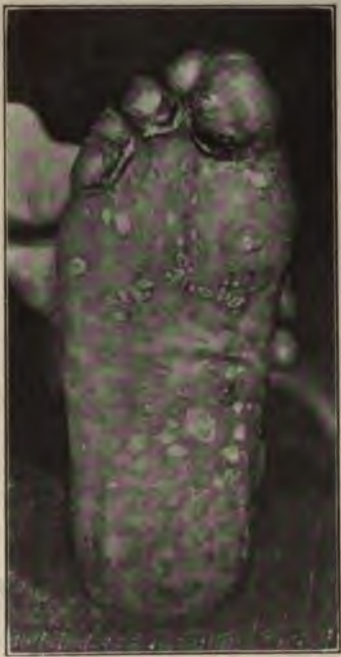


FIG. 147*e*.—PLANTAR SYPHILIDE. Abundant scaling papular eruption upon the sole, mucous patches between the toes. Early secondary period. (Author's collection.)

When these scaling patches occur upon the soles there is apt to be more induration and a good deal more scaling, and they are frequently associated with an abundant eruption of papules between the toes, which at this site, on account of maceration, quickly lose their unhealthy epidermis and become fissured, weeping lesions with an offensive secretion, presenting the characteristics of mucous patches or condylomata.

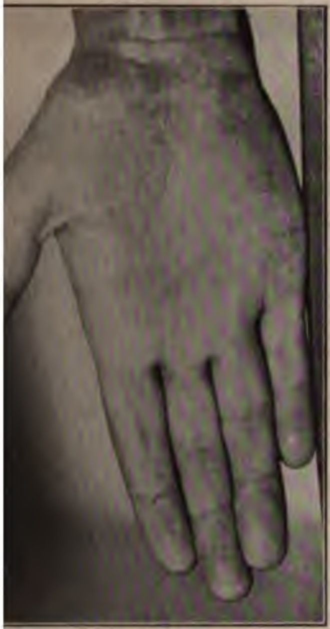
The palmar syphilides are frequent in the secondary period, in association with any of the other secondary syphilides, and in the secondary period are usually symmetrical.

The scaling palmar syphilide is also a common relapsing eruption of syphilis, occurring long after all other evidences of active syphilis have disappeared, and it may be a late tertiary manifestation of the disease. When recurring as a late relapsing eruption it is usually confined to one hand or foot and shows still stronger tendency to circinate configuration. As a tertiary eruption it may ulcerate, but this is not common.

The palmar and plantar syphilides are especially persistent and rebellious to treatment. When occurring as a secondary eruption they disappear slowly under treatment, often being the last evidence of the disease to vanish. The scaling tertiary lesions may persist or relapse for months or even years.

The palmar syphilides occurring as a secondary eruption are usually easily diagnosticated by reason of their association with other syphilitic lesions or from the history or evidence of recent syphilis. They resemble palmar eczema, but differ in their circinate, sharply defined border, the

PLATE III



-Large Flat Papular Syphilide
Palm. (Author's collection.)

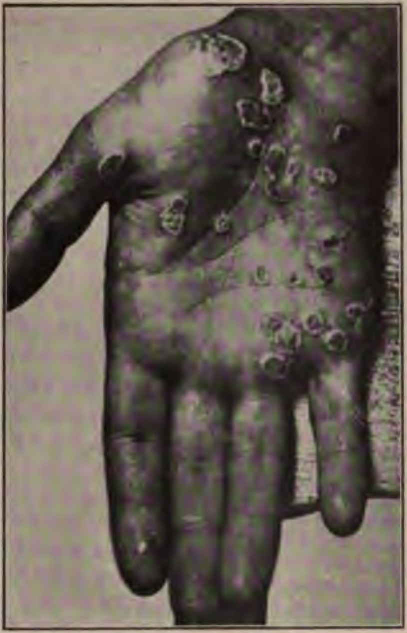
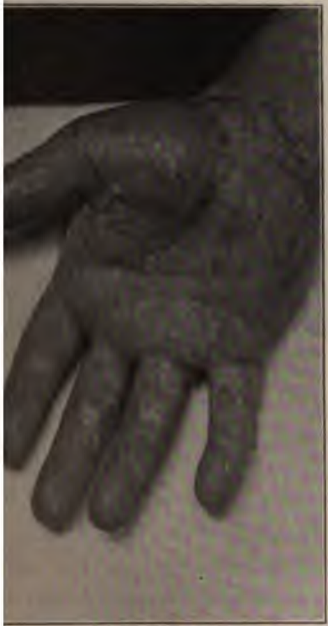


Fig. 2.—Large Scaling Papular Syphilide of Palm. (Author's collection.)



-Diffuse Scaling Papular Syphi-
f Palm. (Author's collection.)

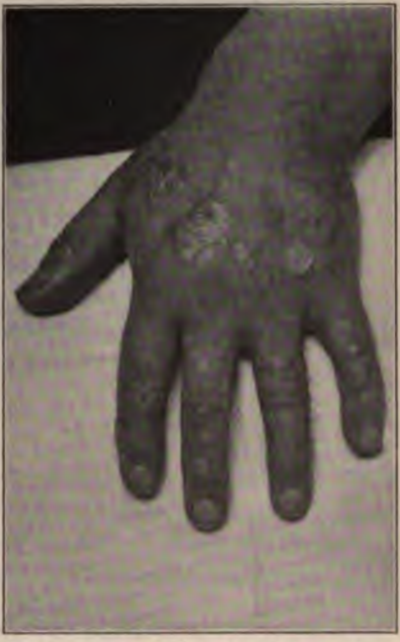


Fig. 4.—Scaling Papular Syphilide of Hand. (Author's collection.)

SYPHILIDES OF HAND

firmer infiltration, their darker indolent red color, and the evidence of greater activity of the process at the border than at the center. The late palmar syphilides differ further from eczema in the fact that they are usually confined to one hand.

Condylomata Lata.—The development of papules about the genitals and anus is common, not only when the eruption is generally papular but in association with all forms of secondary syphilitic eruptions, and from maceration of the lesions at these sites they assume peculiar forms. The epidermis over the papules is thrown off and they are converted into weeping or moist papules. Then the lesions become hypertrophic and form flat, slightly papillomatous, wartlike papules. These individual lesions vary from the size of a pea to a coin, but they are usually coalesced into irregular plateaux from the size of a finger nail to several inches in diameter. These are raised from an eighth to a quarter of an inch above the surrounding skin, are sharply elevated at the border, and present a flat surface composed of short, papillomatous vegetations. They are covered with grayish necrotic detritus and have an offensive seropurulent secretion which is very contagious. Papules may become condylomatous in any location where they are exposed to continual heat and moisture; thus they are seen also in the groins, on the scrotum and the adjacent portions of the thighs, in the axillary and mammary folds, and occasionally about the mouth and between the toes. They develop quickly and persist for weeks or months unless they have in addition to internal treatment careful local treatment. Condylomata about the genitals are usually accompanied by considerable itching, and from the formation of fissures may be painful.

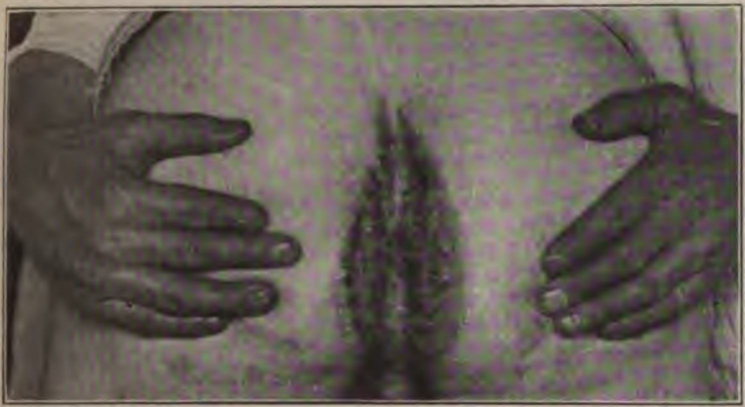


Fig. 148.—Condylomata Lata. (Author's collection.)

They are a characteristic lesion and are likely to be confused only with acuminate condylomata. From these they differ sharply in that they form flat papillomatous plateaux, while the acuminate condylomata occur in sharply raised, conical, filiform, hypertrophied papillæ, that resemble rather minute groups of conical peaks than rough, elevated plateaux. Condylomata lata, from their characteristic appearance and common occurrence even with scant general eruptions, are of great diagnostic importance.

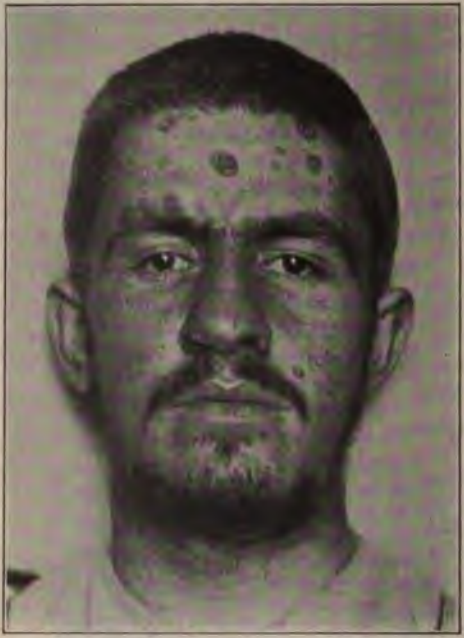


FIG. 149.—FRAMBŒSIOID SYPHILIDE. (Author's collection.)

VEGETATING SYPHILIDE (*Frambœsioid syphilide*).—In some cases the moist papules vegetate abundantly, forming rough, papillomatous, red or grayish, warty overgrowths with abundant mucopurulent discharge. These lesions may develop not only from papules in the secondary stage, but also from any ulcerated or excoriated syphilitic lesion as a result of exposure to moisture and heat and, probably, secondary infection. Their pathology is probably the same as that of other forms of vegetating dermatitis. They are seen not only about the genitals and anus but in the naso-labial fold, the angles of the mouth, and the inguinal and mammary folds. They are uncommon lesions.

PUSTULAR SYPHILIDE (*Acneiform syphilide, Ecthymatous syphilide, Varioliform syphilide*).—Pustular syphilides—and the same is true of vesicular syphilides—are evolved from papules by the occurrence of free exudation in or under the epidermis upon the tops of the papules, and they accordingly present the characteristics of papular lesions. They occur both as flat pustular syphilide, corresponding to the flat papular syphilide, and as follicular or acuminate pustular syphilides, corresponding to the follicular papular syphilide. The pustules develop upon the tops of the papules and quickly dry into dirty yellowish or brownish crusts which soon fall off, leaving slight central ulceration or slight atrophic scarring of the center of the indurated papule, which then undergoes its ordinary evolution. The pustules sometimes form on papules as large as a finger nail, producing large, flat, rather flaccid pustules which dry into yellowish-brown crusts with an eroded or superficially ulcerated base, forming the so-called ecthymatous syphilide. Occasionally there is more marked ulceration of the base forming the pustulo-ulcerative syphilide in which we have finger-nail-sized or larger lesions covered with dirty crusts, under which there is ulceration. These

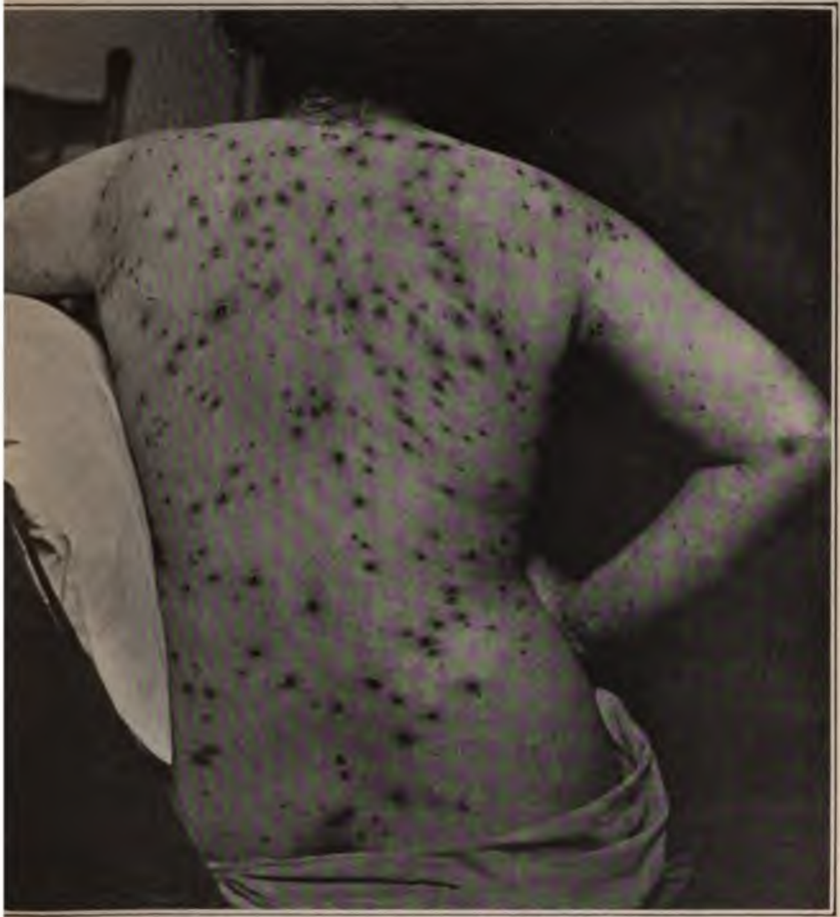


FIG. 150.—PUSTULAR SYPHILIDE. (Author's collection.)

sions are frequently the beginning of rupia or rupial ulcers, to be escribed later.

The acuminate pustular syphilides are situated around follicles, occur n pinhead-sized or larger papular bases, and are often slightly pitted t the tip from the contraction which the canal of the follicle exerts. They quickly dry into small, dirty, yellowish crusts which soon fall off, eaving small atrophic scars.

In distribution, grouping, course, character of the basic infiltration, and absence of subjective symptoms the pustular syphilides resemble the papular. All of them are apt to be followed by more or less scarring.

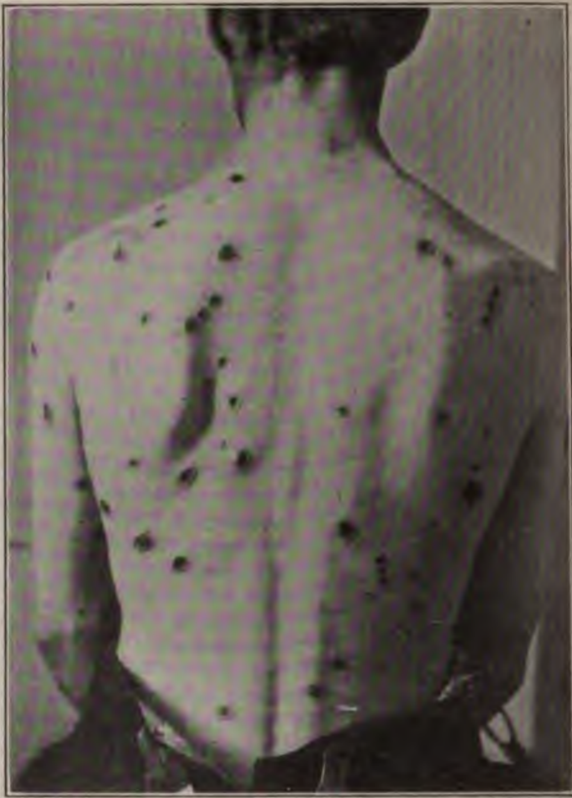


FIG. 151.—LARGE PUSTULAR SYPHILIDE. (Schamberg's collection.)

When the lesions are large and the pustulation is accompanied by considerable ulceration scarring may be pronounced.

Pustular syphilides are a manifestation of severe forms of syphilis, and are usually seen in individuals who are not in vigorous health and who have neglected treatment. This form of eruption, therefore, is seen most frequently in public practice and is rare among private patients who are able or willing to take proper care of themselves.

The pustular syphilides ordinarily occur within the first six or eight months of the disease as a generalized eruption combined with papules, and frequently with ulcerative lesions. They may occur also late in the course of active syphilis as a relapsing eruption.

The diagnosis of syphilis in the presence of pustular syphilides is readily made because of the constant association of other syphi- nd other well-marked evidences of active syphilis. The large ate pustular syphilide may at times possibly be confused with The limitation of acne to the face and the front and back of the half of the chest, the inflammatory character of the lesions, their around dilated sebaceous glands, the presence of many come- along with the inflammatory lesions, the duration of the disease rs, its occurrence during adolescence, and the absence of concomi- idences of syphilis form so clear a picture that the confusion with n result only from careless consideration of the case.

abundant pustular syphilide accompanied by syphilitic fever may nes be difficult to differentiate from variola. Here usually crucial e is given by the characteristic course of syphilis as contrasted at of variola, and the accompaniment of other evidences of syph-

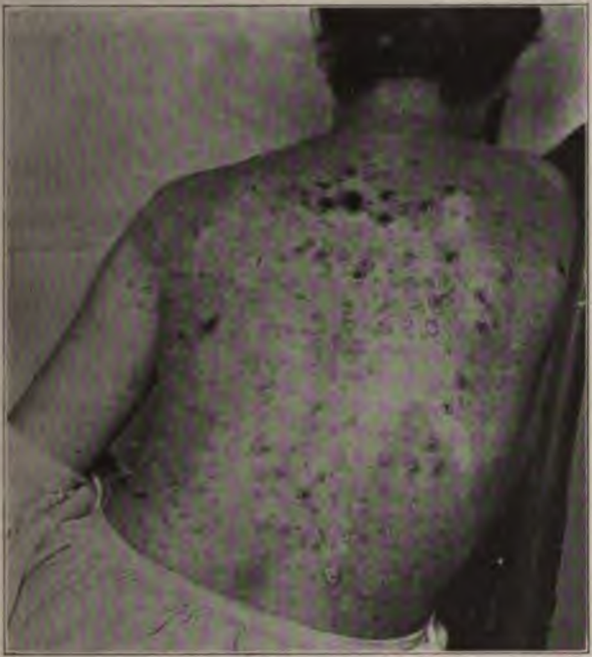


FIG. 152.—ECTHYMATOUS SYPHILIDE. (Author's collection.)

e mucous patches, condylomata, and traces of a recent chancre. r, the pustular syphilide occurs abundantly on the trunk as well as face, while variola is much more abundant on the face. The tic pustule becomes a pustule immediately and does not pass h the characteristic changes of the variola pustule. The lesions how a tendency to circinate arrangement in syphilis; they appear irregularly and they are associated with papular and ulcerative lesions, while the lesions of variola come out in one crop and are uniform.

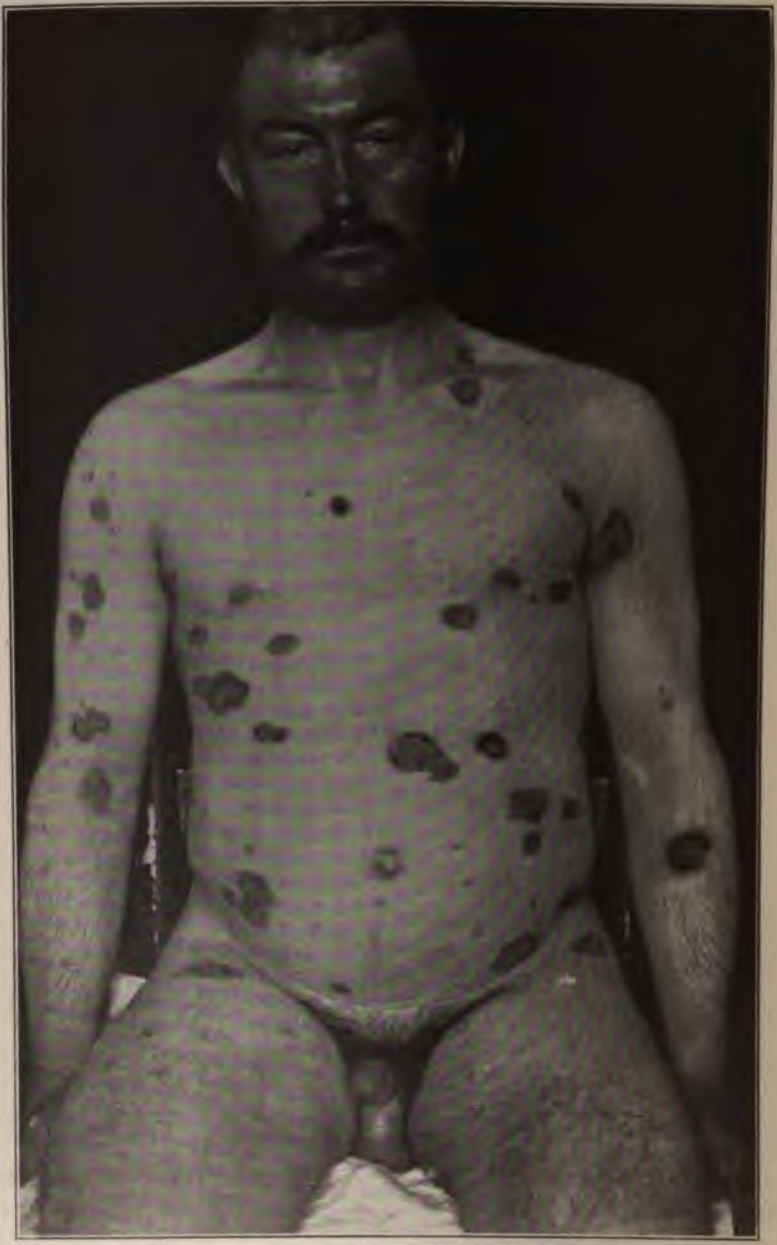


FIG. 153.—ANNULAR PAPULO-ULCERATIVE SYPHILIDE. Secondary period. (Author's collection.)

Bullous or Pemphigoid Syphilide.—Large pustular syphilides may enlarge at the periphery to an inch or more in diameter with the formation of large, not very tense, purulent bullæ—pemphigoid or bullous syphilides. This is an extremely rare lesion in acquired syphilis, but is common in the congenital form. The bullæ have an infiltrated base with more or less ulceration. They quickly subside with the formation of a crust under which healing may take place with very slight ulceration and slight scarring. Sometimes the crust forms over the central part of the lesion and leaves a peripheral pustular ring. Again, the lesion may subside into a crust and ulceration proceed beneath this with the formation of rupial ulcers. These lesions are always associated with other syphilides and other evidences of syphilis.

Vesicular Syphilide (*Varicelliform syphilide, Herpetiform syphilide*).—Vesicular syphilides are much rarer than pustular syphilides, and are a very unusual form of eruption. They are of the same types as the pustular syphilides except for their serous contents, and are always associated with pustular lesions. The small vesicular syphilides correspond in character with the small pustular syphilide. The vesicles may be converted into pustules or may dry up without rupturing in the course of a few days, and form scales and then undergo the involution of syphilitic papules. The large vesicular corresponds to the large pustular syphilide, and is associated with pustular lesions. The lesions undergo the same evolution as the small vesicular syphilide.

Hutchinson has described a bilateral localized vesicular eruption in syphilis which is indistinguishable from zoster except for its symmetry, and Crocker has seen such a case, but it is an excessively rare syphilitic eruption.

Mucous Patches.—In the secondary stage of syphilis it is almost constant for lesions to appear upon the mucous membrane of the mouth. These lesions correspond to the macules and papules upon the skin, but their appearance is altered by the maceration to which they are exposed. They are known as mucous patches and, because of their constant occurrence, are of great importance in diagnosis. They occur upon the mucous membranes as red spots which quickly become abraded, with the formation of round or superficial excoriations which are more or less covered by necrotic epidermis in the form of a grayish or more frequently grayish-yellow pellicle. This is ordinarily closely adherent, but sometimes can be separated, and then beneath it the surface is bright red and bleeds easily. The mucous patches are usually round or oval, from the size of a tackhead to that of a finger nail, with a sharply defined border and surrounded by a narrow red areola. They are without induration. The excoriated surface is usually slightly depressed or level with the surrounding mucous membrane; exceptionally it is slightly elevated.

Occasionally as the result of infection they ulcerate, but this is not frequent. Ordinarily oval or rounded, mucous patches occasionally are evidently made up of groups of small lesions which are arranged in crescentic or even circular figures. They occur most frequently on the mucous membranes near the border of the lips, especially about the angles of the mouth, and they may spread over the vermilion border. Their next most frequent site is the buccal mucous membrane adjacent to the molar teeth, but they may occur upon any part of the oral and pharyngeal mucous membranes.

Their number varies greatly. Frequently only one or two lesions can be found by careful search. In other cases the entire mucous membrane of the lips and the greater part of the mucous membrane of the mouth will be excoriated by them. They occur not only in the mouth, but to a less extent upon the vaginal and anal mucous membranes. They are also found occasionally upon the skin of the folds of the body. They usually develop along with the first eruption of syphilis, and recur during the active period of the disease. Often they are the last eruptive evidence of the disease to manifest itself. Ordinarily they are more or less persistent, but on the whole the condition is kept up in the mouth, not by the persistence of individual lesions, but by the constant development of new lesions.

Purpura.—Extravasation of blood into the skin producing purpuric patches on the legs has been observed in secondary syphilis, independent of the purpura which is sometimes produced by the use of potassium iodide. It may be associated with hæmorrhage of the mucous membranes. It is a very rare occurrence in acquired syphilis, but is somewhat less infrequent in congenital syphilis.

Combinations of Syphilides with Other Skin Diseases.—Syphilides may occur in combination with almost all diseases of the skin. In such cases ordinarily the diseases pursue their independent courses with only such results from the symbiosis as are inevitable from the presence of two eruptions upon the same surface.

The influence of infection with the common pus organisms is seen constantly in ulcerative lesions of syphilis, especially in the uncleanly and very poor. But this secondary infection does not, as a rule, influence the character of the lesions to any essential extent. A complication which is common with syphilides, especially papular and macular, arises from the presence of seborrhœa or seborrhœic dermatitis on the face. It is the more frequent because syphilis itself exaggerates or excites seborrhœa. When this combination occurs the appearance of the syphilitic eruption is somewhat altered. It becomes of a duller red color, greasy, with the formation of more or less greasy, yellowish scales, and the outline of the lesions becomes less well defined on account of an ill-

ned periphery of inflammation, the result of the seborrhœic derma-
. The formation of greasy scales may be quite free, and is most
ndant in the usual locations of seborrhœic dermatitis, in the naso-
l folds, upon the glabella, and on the eyelids.

'igmentary Changes in Syphilis (*Pigmentary syphilide, Syphilitic
derma*).—All syphilitic lesions are apt to be followed by temporary
entation at their sites from the deposit of blood pigment in the tis-

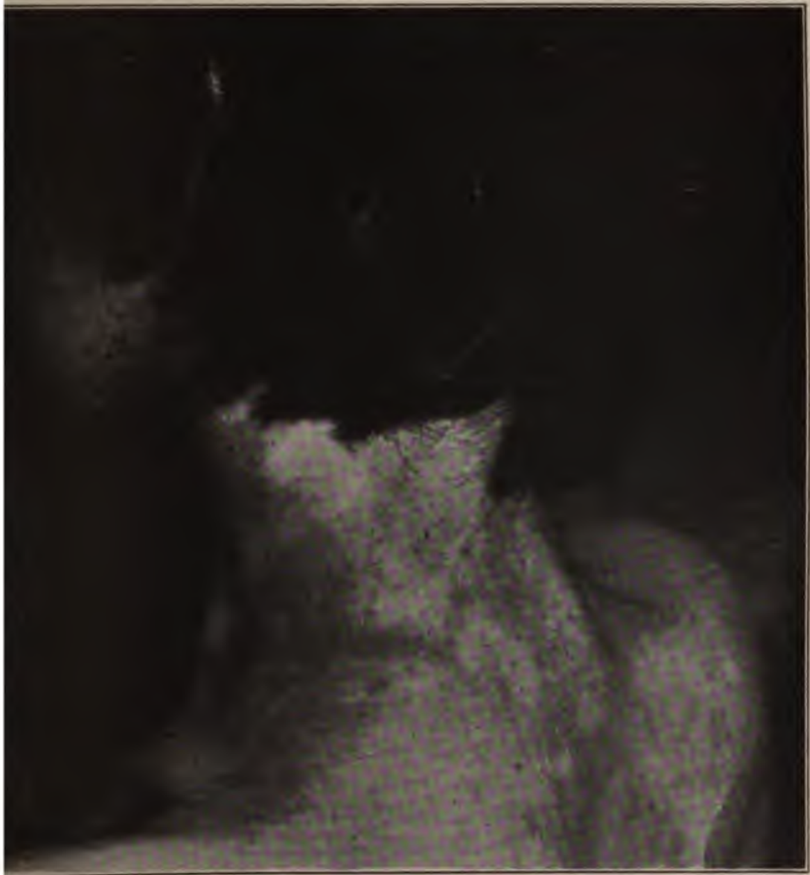


FIG. 154.—SYPHILITIC LEUKODERMA. (Author's collection.)

. After syphilitic ulcerations there are also pigmentary changes.
scars which follow are at first pigmented, or red with an areola of
nentation. Ultimately these scars usually become permanently white
are surrounded by a pigmented areola which finally fades out; on
legs it may be permanent. These pigmentary changes are secondary
he pathological processes producing the ordinary syphilides. In addi-

tion there is a true pigmentary disturbance which is occasionally seen in the course of secondary syphilis. It consists in the formation of roundish patches the size of a finger nail or larger, which are whiter than normal from the loss of pigment, while outside these patches there is increase of pigment so that the white spots are shown in greater contrast. Unlike ordinary leukoderma the transition at the border from infrapigmented to hyperpigmented skin is gradual, and the borders of the patches are accordingly rather ill defined. These patches occur symmetrically on the back and sides of the neck and produce a retiform mottling of the skin of the invaded area. This disturbance of pigment is found almost exclusively upon the back of the neck, sometimes upon the shoulders, and as a rule in dark-skinned women. It usually develops during the second six months of active syphilis, and is persistent, lasting for months or a year or more, and is slightly if at all influenced by treatment.

There is some difference of opinion as to whether this is a true pigmentary disturbance or is the result of preceding ordinary syphilides. It undoubtedly occurs independently of any preceding syphilide. Its location and the peculiarities in size and distribution of the lesions are sufficiently characteristic to demonstrate its independence of ordinary leukoderma and its essential relationship to syphilis.

Taylor mentions disturbances of pigmentation which consist of various-sized brownish spots or diffuse patches of increased pigmentation without any leukodermatous lesions. Apparently they are analogous to chloasma. They must be extremely rare.

Involvement of the Appendages of the Skin.—Temporary alopecia is a common, and onychia an occasional, occurrence in secondary syphilis.

ALOPECIA.[1]—The alopecia which occurs is analogous to that which is frequently seen in acute infectious fevers, and is doubtless of toxic origin. It may occur either as a diffuse thinning of the hair or as an irregular thinning in patches from half an inch to an inch in diameter. The diffuse thinning occurs more or less over the entire scalp, but is most pronounced at the sides and back. The changes simply result in a diminution in the total quantity of the hairs. This diminution may be only slight; in many cases it is so abundant that the pale scalp is readily seen through the remaining hairs. The more common form is the irregular shedding in roundish, thumb-nail-sized patches. These do not become entirely bald, but the hair becomes greatly thinned over them so that the scalp can be seen. The borders of the thinned patches are ill defined and gradually merge into the surrounding more hairy areas. These patches occur in numbers close together and symmetrically on the scalp, and produce a mottled thinning of the hair of so-called moth-eaten appearance which is quite characteristic. This alopecia is most marked upon the back of the scalp and to a less extent upon the sides. In either form of alopecia the hair which does not fall out becomes lusterless and dry. In addition to loss of hair of the scalp there may be loss of hair upon other parts of the body, but this is not frequent.

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[1] Klotz, *Trans. Am. Derm. Assn.*, 1906.

Syphilitic alopecia develops rather rapidly; the hair falls out so freely in combing or rubbing the scalp that the patient is alarmed lest he become bald, but complete baldness even in circumscribed patches rarely, if ever, occurs. Syphilitic alopecia may disappear rapidly under treatment and not persist for more than three or four months, but it is likely not to disappear entirely for a year or more. It is sometimes the precursor of permanent baldness, but as a rule the return of hair takes place as in alopecia resulting from other infectious diseases.

Of course, after syphilitic ulceration upon the scalp there may be permanent baldness in the scar tissue from destruction of the hair follicles, but this type of circumscribed baldness is in no way peculiar to syphilis.

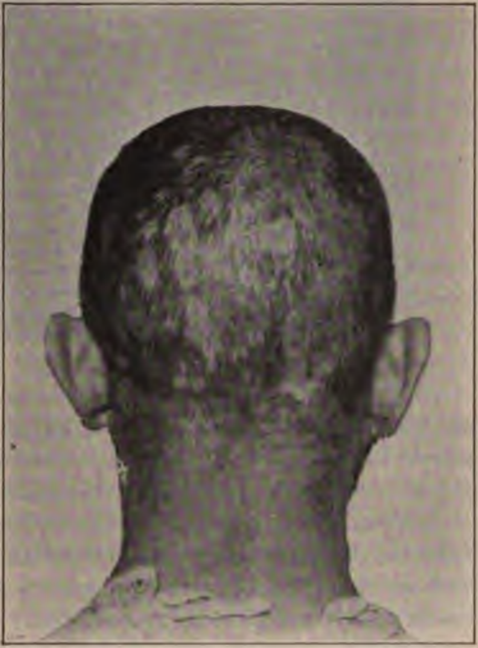


FIG. 155.—SYPHILITIC ALOPECIA.
(Author's collection.)

NAILS.—The nails may, without the occurrence of definite syphilitic infiltrations about them, become opaque, lusterless, and brittle, with a tendency to break at the free edges, and show furrowing or worm-eaten irregularities. These nail changes are the result of the disturbance of the general nutrition, and are apparently analogous to the changes produced in the hair. They are also analogous to the nail changes which are seen occasionally in the course of many constitutional diseases, such as the acute infectious diseases, and of disturbances of metabolism, like gout.

ONYCHIA AND PARONYCHIA.—The nails may also become involved from the development of syphilitic infiltrations in the nail bed or in the skin near the nail, producing onychia or paronychia. In syphilitic onychia papules develop in the nail bed under the nail and they may break down with ulceration. The nails themselves become thickened, opaque, and friable, and marked by transverse furrows or irregularities. They are occasionally entirely detached, leaving the syphilitic infiltrations exposed. When this occurs a new nail slowly grows; this is at first thin and abortive, and is likely to be permanently deformed.

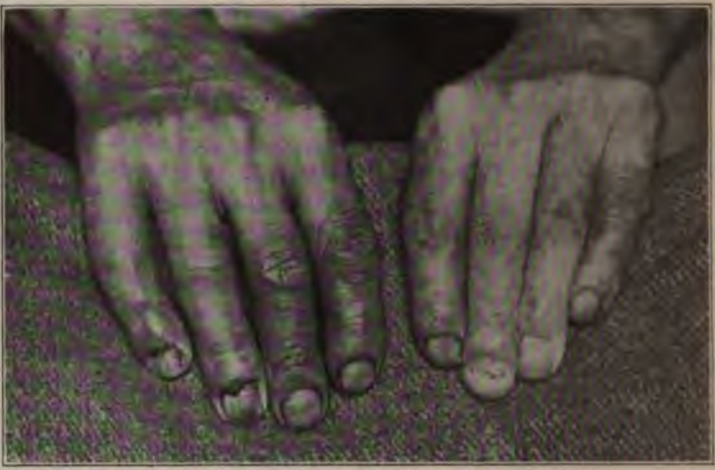


Fig. 156.—Syphilitic Onychia. (Author's collection.)

Similar changes in the nails may occur from paronychia—that is, from the development of syphilitic infiltrations in the skin near the nails, and their subsequent spread to involve the subcutaneous tissue in the borders of the nails or in the nail bed.

Syphilitic onychia and paronychia are seen in secondary syphilis at any time during the first year, and are usually an accompaniment of abundant papular or pustular eruptions. They are rather persistent and are slow to disappear. It is occasionally more than a year before the nails regain their normal or approximately normal condition. Occurring in secondary syphilis the condition usually involves several or all of the fingers, and may also involve the toes.

Both onychia and paronychia may occur in late syphilis from the development of gummata in or around the nail bed. In these cases the mechanism of the production of the lesions is the same as with secondary lesions—that is, the nail disturbance is secondary to a syphilitic infiltration involving the subcutaneous tissue of the nails. But when these lesions occur in tertiary syphilis they are usually confined to one hand or foot, and involve only one or a few nails of the affected extremity.

Tertiary Lesions.—The so-called tertiary lesions of syphilis are all essentially of the same character; they are gummata, infective granulomata, which show the features emphasized by Kaposi. They are not able to undergo organization into permanent connective tissue, and they undergo resolution from the center, so that in resolving lesions the borders show the most active part of the disease.

As already indicated, gummatous lesions may occur at any time during the course of syphilis, but they appear only exceptionally in the secondary stage, and as a rule develop after all manifestations of active general infection have disappeared. Their appearance is possible at any time in the subsequent life of the syphilitic patient, but the chances of their development diminish directly with the length of time which has elapsed since the active stage of the disease. In the majority of treated cases tertiary lesions never occur. When they occur after the active period of syphilis has passed they are not contagious except in the rarest instances; they still, however, have the essential characteristic of syphilitic lesions of yielding to antisyphilitic treatment.

In contrast with the lesions of the active period of syphilis tertiary lesions occur in local foci and not as generalized eruptions. Lesions may, however, occur in several or many locations. Ordinarily they show no symmetry. A tertiary eruption may occur as a single lesion but, except in the case of large nodules, the lesions are most frequently multiple. The multiple lesions occur in groups and show a marked tendency to circinate arrangement. They tend also to spread peripherally with the formation of serpiginous ulcers, to be referred to again shortly.

Syphilitic Scars.—Scarring frequently occurs upon the resolution of nonulcerative tertiary infiltrations, especially if they are in the skin instead of beneath it. It always occurs from the ulcerative lesions, and this scarring is characteristic. The scars of ulcerating syphilides are as a rule atrophic and covered by smooth skin, without the usual papillary markings of the skin, but frequently minutely pitted at the follicular openings. They often look as though the upper part of the corium and papillary layer had by some device been dissected out from under the epidermis, and the epidermis allowed to drop down upon the smooth surface beneath. Frequently the skin is so thin and pliable that it is thrown up into minute papery wrinkles, giving it an appearance which has been likened to cigarette paper. When ulceration has been deep, the scars, instead of being of the thin atrophic character, may be thickened and keloidal at the center, but in such cases the typical atrophic character of the scarring is usually manifest toward the border. At first the scars which follow syphilitic nodules are dark red or purplish, surrounded by a purplish or pigmented areola. Gradually the congestion which causes this color disappears, and the scars are then of a dead or ivory-white color, around which the pigmented areola persists for months, ally ultimately disappears. When the scars are about the lowe the legs they are apt to be permanently pigmented. As r of infiltrations takes place and the scars become healthy, no evi active disease will be found in the scars and new infiltrations p never develop in them. This is a point of great diagnostic im in differentiating ulcerating syphilitic lesions from lupus and epi in which recurrence of the disease in the scars is the rule. T acteristics of the scars referred to above are, of course, most ma the scars of large areas, but they belong as well to the scars smallest tubercles.

Varieties.—In spite of the essential identity of all of the eruptions of tertiary syphilis, they occur in several forms, whic for their differences upon the site in the skin of the infiltration, and number, their arrangement, and their course. Two types o lesions are clinically distinguishable, the tubercle and the gum distinction between these is partly one of size, the tubercle small nodule and the gumma the larger one, but the more impo tinction is that the tubercle is primarily an intracutaneous lesi the gumma is subcutaneo tubercles and gummata multiple. The number is as a rule in inverse p to their size, the tubercl ring in groups of severa the largest gummata us ing single.

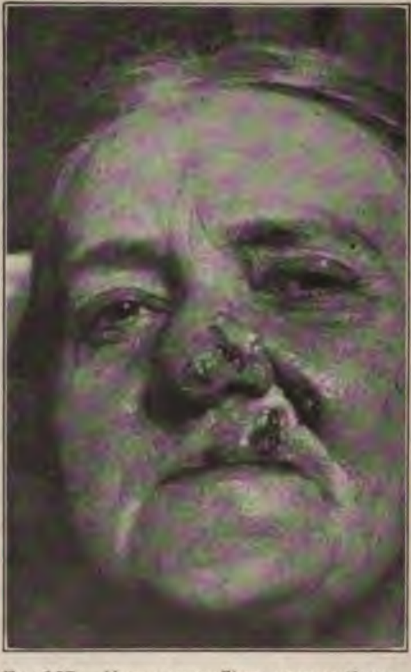


FIG. 157.—ULCERATING TUBERCULAR SYPHILIDE. Tertiary period. (Author's collection.)

TUBERCULAR SYPHIL *ular syphilide*).—Tubercu ilides are cutaneous Individual lesions close ble the large papular of the secondary stage, the secondary syphilide resolve without ulcerat tendency to ulceration in tiary syphilide is strong.

Syphilitic tubercles a infiltrations from an half an inch in diame well-defined borders, imb the skin but usually abruptly from the surfa are rounded or flattened in contour, firm, tense, smooth, or rarely scaly, and of dark red, ham color, or purplish. They usually occur as groups of several or many lesions, making up a localized focus of disease. Occasionally they are widely disseminated; this eruption, however, is not usually regularly diffused, but consists of circumscribed patches. They may occur upon any part of the body, but they show a predilection for the face, especially about the nose, the forehead, the ears, the back of the neck and shoulders, the back of the arms about the elbows, and the legs, especially the outer surface of the thighs. They are essentially indolent in character, and are ordinarily painless except as result of irritation or traumatism. They are very persistent and slowly destructive, and they may spread over wide areas. Ordinarily, however, after reaching a certain size they tend to spread but little farther and may gradually but very slowly undergo resolution.

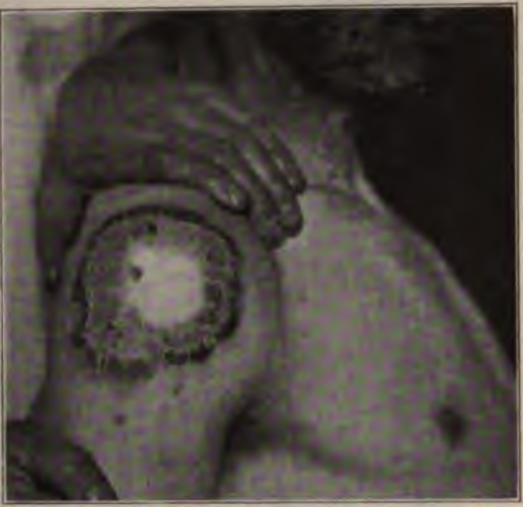


FIG. 158.—ANNULAR SERPIGINOUS SYPHILIDE. Note subsidence in intensity toward center where thin white scarring has already appeared. (Author's collection.)

They may remain as dry lesions throughout their course and ultimately undergo resolution without ulceration. In such cases the nodules flatten down, become softer, and finally disappear, leaving pigmented spots and usually white depressed scars at the sites of the tubercles. More frequently they ulcerate. In an ulcerating tubercle there will be found the characteristic syphilitic border, hypertrophic, firm, dark red, or purplish, with the top excavated by an abrupt ulcer. From the ulcer there is a yellowish, gummy discharge which dries into dirty grayish or greenish-black crusts. The firm, raised, infiltrated, purplish border with deep central ulceration forms a characteristic picture from which the expert can usually make a definite diagnosis of syphilis. As the nodules are grouped the shape of the ulcers as a whole varies very much.

Frequently the tubercles will occur in an irregular group, as, for example, is frequently the case about the tip and alæ of the nose. Such a lesion may show irregular ulceration corresponding to the individual ulcerating tubercles or may, from the coalescence of the smaller ul form a continuous ulcer which has an irregular or circinate outline is perhaps covered by an accumulation of dirty grayish or blackish cr Upon removal of the crusts the character of the lesion will be proclai

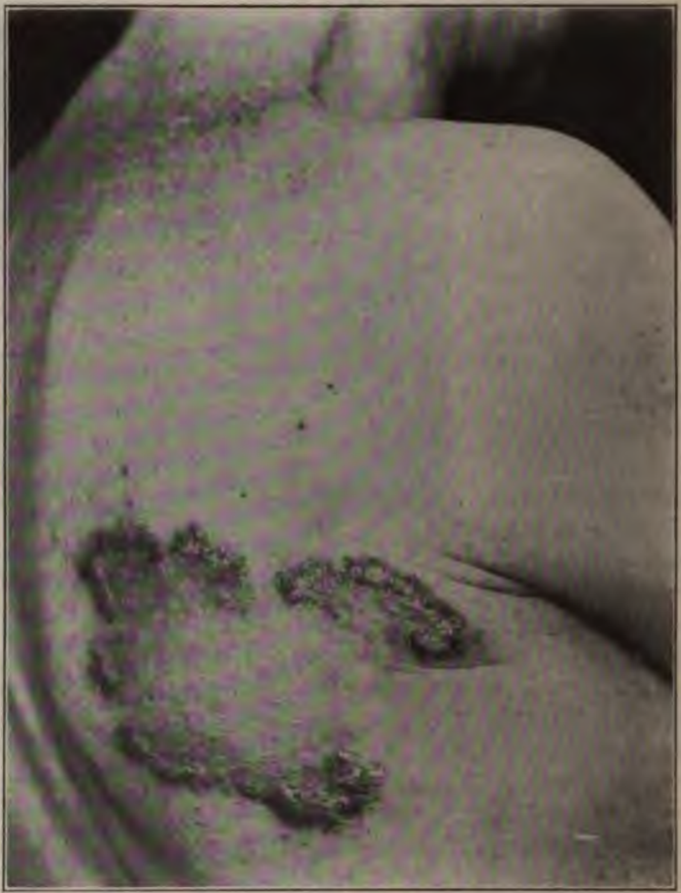


FIG. 159.—SERPIGINOUS ULCERATING SYPHILIDE. Late tertiary period. (Author's collection.)

by the peculiar, rolled, firm, syphilitic border, and almost surely als discrete satellite tubercles of characteristic appearance around the der. Occasionally, as in the papular secondary eruption, the tuber develop in corymbose groups and corymbose ulceration may take pl but this is not frequent.

As a rule the tubercles are arranged in a crescentic group, usu forming an arc, sometimes a complete circle. In this case when ulc tion occurs it will produce a curved ulcer corresponding to the arra ment of the tubercles. If the group consists of only a few tubercles, there will be formed a kidney- or bean-shaped ulceration; if of many tubercles, the outline will be crescentic or horseshoe shaped, or perhaps annular. In these circinate figures there is a marked tendency for the disease to spread peripherally, causing so-called *serpiginous ulceration*, which is characteristic, if not quite pathognomonic, of syphilis. The spreading may take place by continuity from a curved line of infiltration, but more frequently it occurs by the development of fresh ulcerating tubercles at the periphery. Thus there will be formed a serpiginous

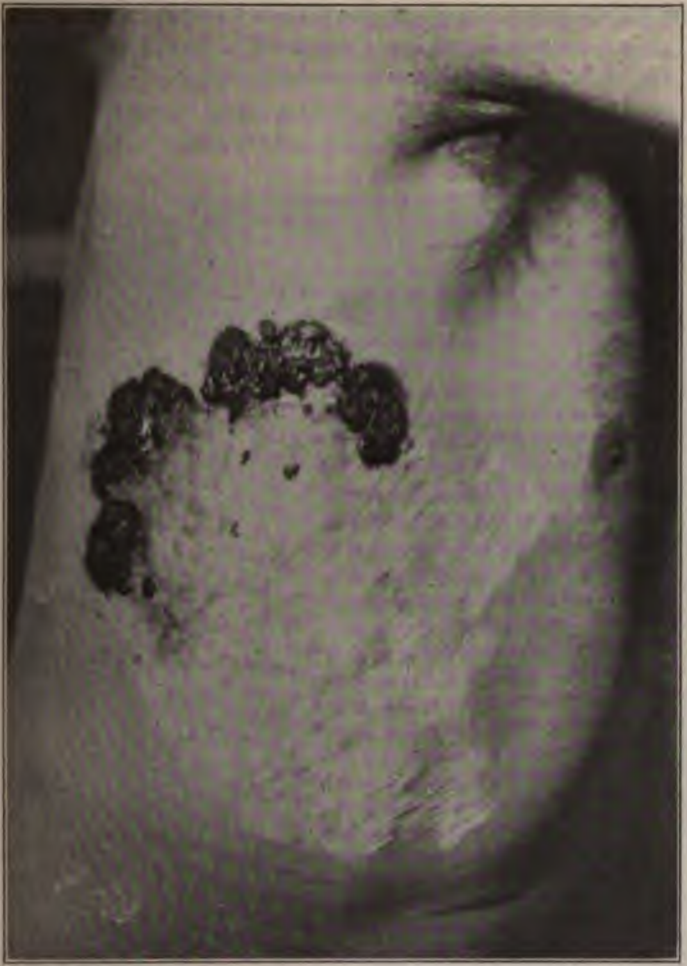


FIG. 160.—SERPIGINOUS ULCERATING SYPHILIDE. Note thick rupial crusts over ulcers, and cigarette-paper scarring in the area over which the ulceration has traveled. (Author's collection.)

ulcer consisting of a curved line of ulceration, active and spreading on the convex border but undergoing resolution on the concave border, and leaving in its track superficial thin scars. The border of active disease may be composed of discrete ulcers corresponding to single tubercles, or there may be a crescentic ulcer the width of one or two tubercles and continuous throughout the whole curve of infiltration. Again, there may be in the border irregular-shaped ulcers the size of a coin, or larger from the coalescence of adjacent tubercles. The disease may spread slowly for months, and gradually traverse large areas, leaving in its track fan-shaped or rounded scars of large size. Such scars rarely cause distortion, because there is little connective tissue in them to contract.

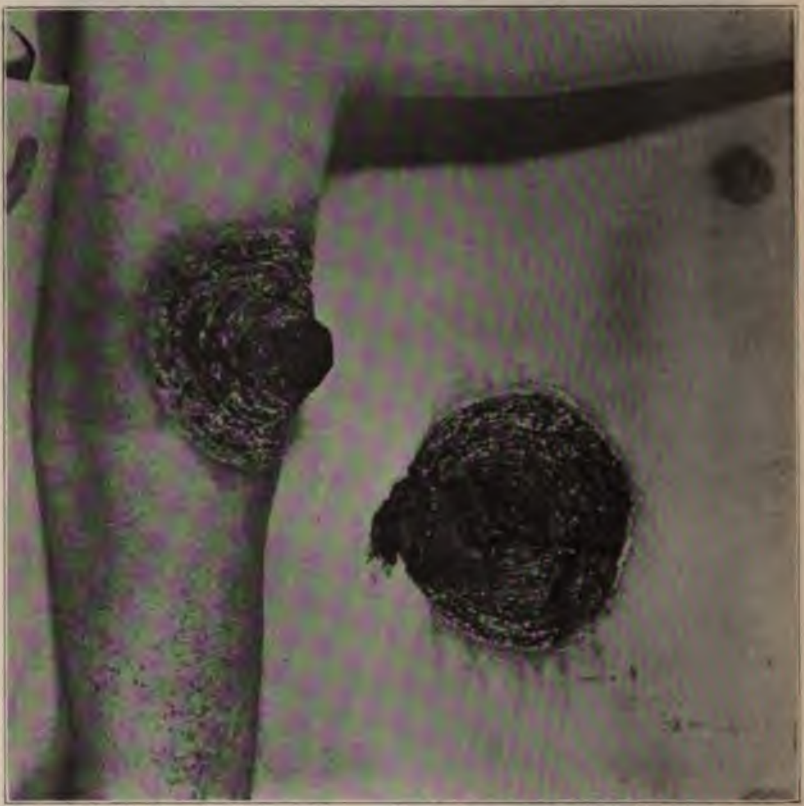


FIG. 161.—SYPHILITIC RUPIA. Unusually regular in formation. (Whitehouse's collection.)

RUPIA.—Individual tubercles or groups of tubercles may ulcerate with the production of heaped-up, laminated crusts, which characterize the lesions denominated as rupia or rupial ulcers. The mechanism of the production of rupia is as follows: there occurs in the skin a nodular or more frequently a flattened infiltration the size of a pea or finger nail. This ulcerates at the center; the secretion dries into a dirty yellowish or blackish crust; under this crust the secretion then accumulates, and at the same time the ulcer spreads peripherally. In this way a second and somewhat larger crust is formed beneath the first. The process continues and a third crust is formed. By this time there will be an ulcer covered by a crust composed of three laminæ, each somewhat larger than the one above, and so the process will continue with the production of a laminated crust perhaps a quarter to a half an inch or more thick. This crust is roughly conical, laminated, and presents somewhat the appearance of a blackened oyster shell. Underneath it there will be the unhealthy ulcerating surface of a broken-down syphilitic nodule.

These rupial ulcers may form also from large flat pustular or bullous syphilides. In that case the contents of the bulla dry into a crust, and the further formation of the rupial crust is the same as in an ulcerating tubercle. These lesions are characteristic and are common lesions in ulcerating syphilides.

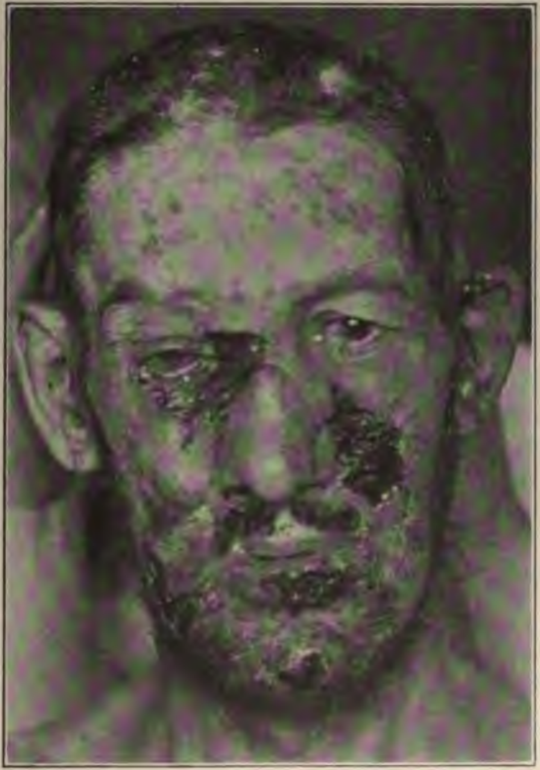


FIG. 162.—ULCERATING GUMMATA. Late tertiary. Note characteristic white scars on forehead and scalp. (Author's collection.)

THE GUMMATOUS SYPHILIDE.—The larger subcutaneous syphilitic nodules are of the same character as the tubercles already described, and go through the same evolution. Like the tubercles, they may undergo fatty degeneration and absorption without ulceration, but more frequently they break down with the formation of ulcers. A gumma begins as a subcutaneous nodule the size of a pea or bean or larger. At first it is not elevated, but can be felt as a firm, elastic, painless nodule beneath the skin.

From this size it enlarges to the size of a hazelnut or olive or walnut. Occasionally it may increase into a tumor the size of an egg or an orange, and Fournier has recorded a gumma 14 cm. in length by 10 cm. in breadth and 2 to 6 cm. in thickness. In shape gummata may be oval or globular or flattened disks. Ordinarily when fully developed they project more or less above the skin, but when deep seated or diffuse they may be without elevation. At first they do not involve the skin, and it is movable and of normal color. Later, as they enlarge, the skin becomes involved in the tumor and of a reddish or purplish color. From this point they may disappear by absorption after persisting for weeks or months. More frequently they ultimately become cyanotic, boggy, and fluctuating, and break down at the center. Sometimes the openings are multiple, but this is not the rule. The ulceration which results from their breaking down depends upon the depth of the gumma. If the gumma has been superficial and spread out into a flattened plaque, there results a relatively superficial flat ulcer of perhaps one eighth to three eighths of an inch in depth. If the gumma has been deep there results a deep, sharply excavated, roundish ulcer. The border of the ulcer is thickened, purplish, usually indurated and firm, perhaps flabby. The walls are undermined and irregular, the floor uneven and covered with necrotic tissue, and there is a secretion of gummy, seropurulent fluid, mixed with particles of sloughing tissue, which dries upon

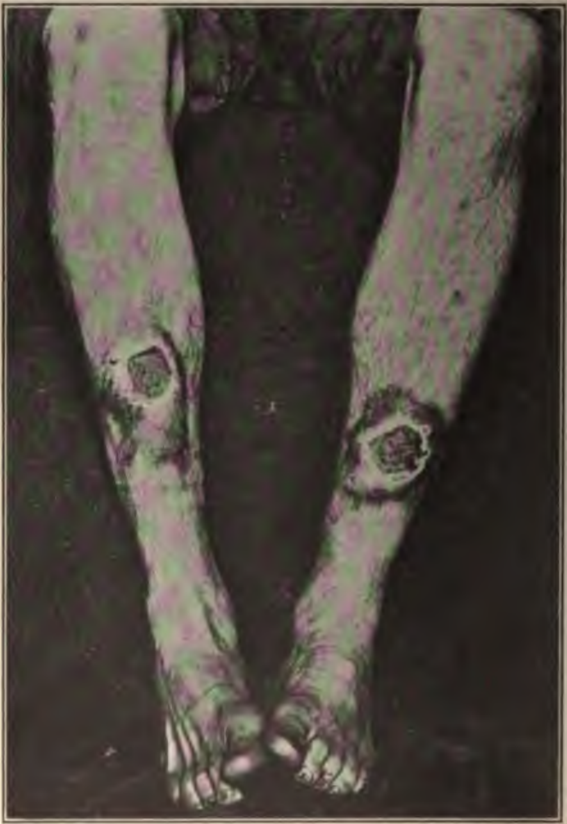


FIG. 163.—GUMMATA. Symmetry unusual. (Author's collection.)

the surface into a dirty blackish crust. Occasionally such an ulcer shows a tendency to serpiginous spreading in the same way that other tertiary lesions do.

The course of such an ulcerating gumma is indolent and slow. It may persist with little or no spread or destruction of tissue for weeks or months, and ultimately undergo spontaneous resolution. Under treatment such lesions heal rapidly. In healing the broken-down and necrotic tissue in the ulcer is thrown off, granulations first cover the base and edges, then fill the cavities, and finally cicatrization takes place. The scar from a gumma is usually relatively small in comparison with the size of the ulcer. It is depressed at the center, white, and surrounded by a pigmented border. On the legs the entire surface may remain pigmented. The contour of scars of multiple gummata depends of course upon the contour of the ulcer. If the ulceration has involved fixed structures, like bone, the scar is likely to be adherent.

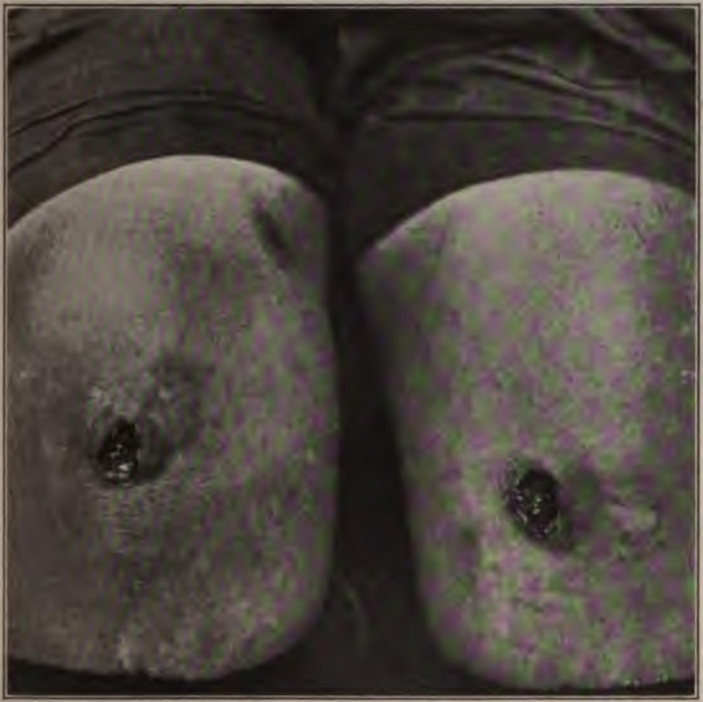


FIG. 164.—GUMMATA OF KNEES. Symmetry unusual. Note scarring where lesions have healed while the infiltrations have spread in front. (Author's collection.)

Usually the number of gummata is few, from one to six or more; occasionally they may be numerous—160 in a case of Lisfranc's. As a rule, their number is in inverse ratio to their size. By the coalescence

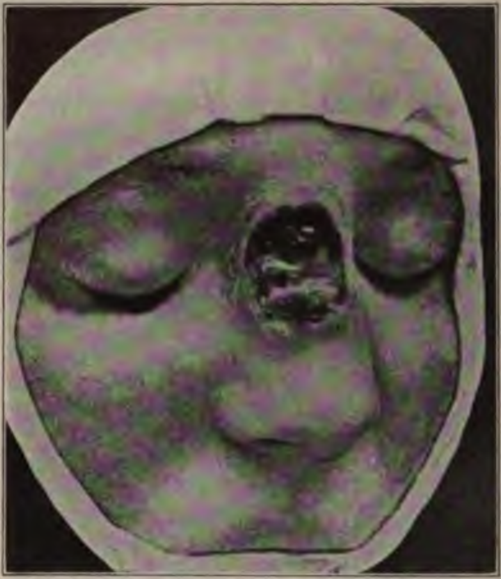


FIG. 165.—ULCERATING GUMMA OF NOSE. (Reproduction in black and white from Jacobi's dermachromes. By permission of The Rebman Company.)

of nonulcerative gummata large tumor-masses may be formed, and by the coalescence of ulcerating gummata large irregular ulcers of any size or shape. Most frequently when ulcerative gummata are numerous they are discrete and form diseased areas in which there are numerous islands of ulceration separated by healthy skin or scar tissue. As a rule, multiple gummata show no symmetry, but occasionally lesions will be symmetrically distributed. They occur most frequently upon the lower extremities, especially the upper two thirds of the legs, particularly just below the patellæ. They are perhaps next most frequent upon the face, especially the forehead, and upon the scalp. They may, of course, occur upon any site, although the palms and soles are almost never involved. The extent of involvement of tissues in gummata knows no limit. They attack muscle, cartilage, and bone, and thus in extreme cases produce great destruction.

As a rule, gummata are characterized by little or no pain except when they are exposed to traumatism or undue motion. Exceptionally, as on the scalp and about the ankles, they may be quite painful. They are unaccompanied by any definite constitutional symptoms, but extensive multiple ulcerating gummatous lesions are frequently associated with marked cachexia.

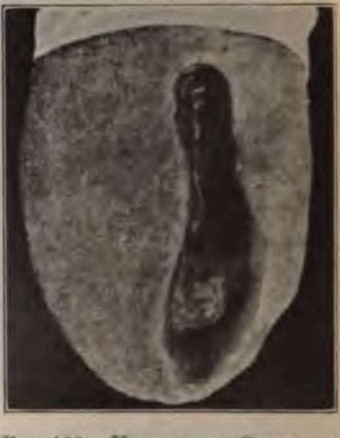
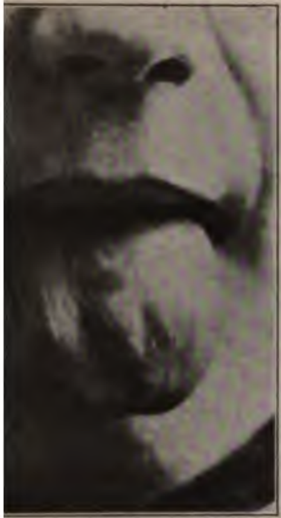


FIG. 166.—ULCERATING GUMMA OF TONGUE. (Reproduction in black and white from Jacobi's dermachromes. By permission of The Rebman Company.)

Phagedena.—As in other ulcers, phagedena may occur in syphilitic ulcers of any period from the chancre on. These ulcers are the result of mixed infection ılent type in cachectic individuals reduced from alcoholism, or other causes which greatly lower resistance. The phag-



ia.—Gumma of Tongue. r W. Wende's collection.)

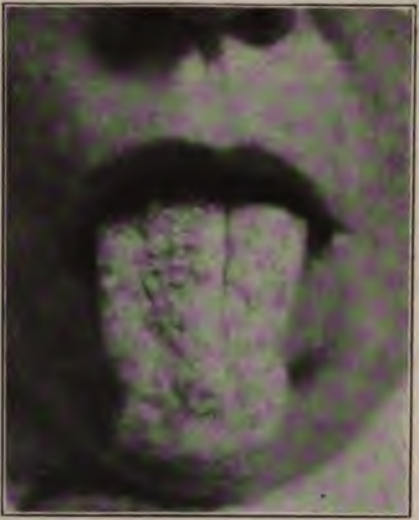


Fig. 166*b*.—Gumma of Tongue. Toad-back variety. (Grover W. Wende's collection.)

s not primarily a part of syphilis, but is an epiphenome- e to secondary infection of syphilitic ulcers in vulnerable ıals.

Congenital Syphilis

ongenital or hereditary syphilis is meant syphilis which is ac- by the fœtus *in utero*. Except for the initial lesion, congenital may present any of the manifestations of the acquired disease. however, certain peculiarities which have to do chiefly with the intensity that the disease usually shows, and with the profound nces of nutrition that it causes. The cutaneous lesions them- resent no material differences from those of acquired syphilis.

hilis may be fatal to the fœtus *in utero;* or the active disease elop *in utero* and the child be born syphilitic; or the child may apparently healthy and develop syphilis within a few weeks after The later the active manifestations of syphilis appear in con- syphilis the milder, as a rule, is the course of the disease.

genital syphilis usually develops within four to eight weeks after nd it is extremely unlikely that the disease will begin later than ths after birth. In a small proportion of cases of hereditary , syphilitic or distrophic lesions may develop several years after ven in the absence of early manifestations of the disease (*syphilis* *hereditaria tarda*, of the French). Of these late manifestations, aside from gummata, interstitial keratitis is the most characteristic. Syphilis hereditaria tarda occurs most frequently between the eighth and twelfth years; it may occur earlier or as late as the thirtieth year (Taylor).

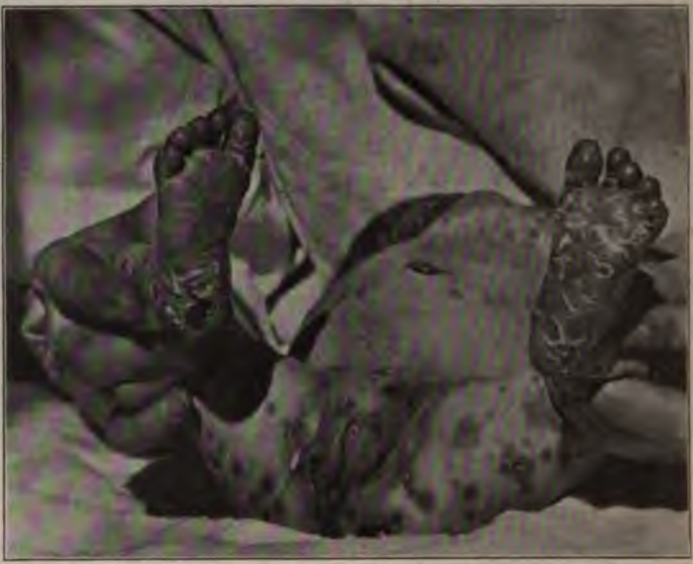


FIG. 167.—HEREDITARY SYPHILIS. (Author's Collection.)

Symptomatology.—A child born with active syphilis is small, cachectic, old looking, and there are apt to be evidences of syphilitic infiltration in many tissues. The spleen and liver are usually enlarged; the bones may be involved, and the epiphyses perhaps separated. In the nose there is rhinitis from ulcerative lesions, with seropurulent or bloody discharge, snuffles, and perhaps complete occlusion of the nostrils, so that the child nurses with difficulty. In the mouth there are mucous patches, the lips are excoriated, and around the angles there are radiating ulcerated fissures. Around the anus and genitals mucous patches may be found, moist papules are common, and usually there are fissures radiating from the anus. One cutaneous manifestation consists of diffuse erythematous patches, especially marked about the buttocks and genitocrural folds. Commonly in these cases there are on the hands and feet and about the wrists and ankles flaccid, purulent, bullous syphilides, always an evidence of a severe form of syphilis. Children born with active syphilis usually succumb in from a few days to a few weeks.

If the child is born infected, but without active evidences of the disease, it is usually healthy looking, well nourished, and without abnormality. Nothing unusual develops immediately, but in the course of four to

ks, rarely later, the disease appears. Its appearance may be
y a period of restlessness and wakefulness, but the first posi-
festation is usually syphilitic rhinitis, which causes snuffles
eres with nursing. The cry becomes harsh and high pitched,
us patches, fissures, and condylomata develop. With the appear-
e active evidences of disease impairment of nutrition and ca-
idly develop. The spleen is usually enlarged, the liver fre-
Occasionally hæmorrhages occur from the mucous orifices,
rom the navel in early cases, and as extravasations into the
e evidences of gummata in various locations may manifest
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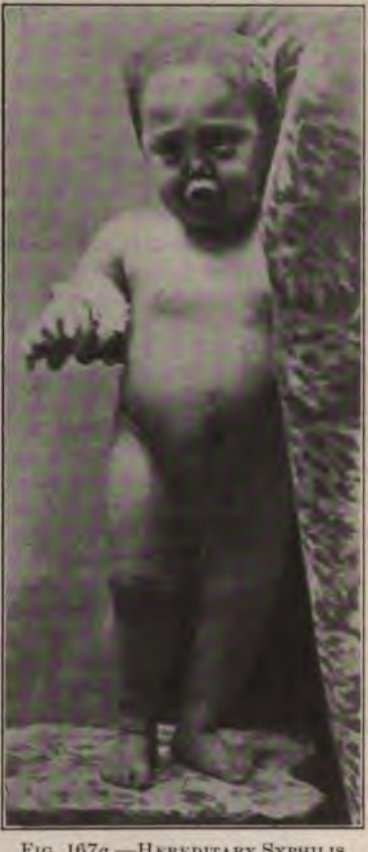

FIG. 167*a*.—HEREDITARY SYPHILIS. (Grover W. Wende's collection.)

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rms of congenital syphilis gummata are apt to occur in the

viscera early in the disease, but in the cases that survive the first few months gummata in the skin are not seen as a rule until after the first year.

Cases which survive the active period of congenital syphilis may later show no manifestations of the disease. Usually, however, the children do not do well, and are small, wizened, and old looking, and at the period of second dentition or at puberty evidences of perverted nutrition are apt to appear. Their growth is slow, and they may show the evidences of infantilism. The forehead is apt to be prominent, the frontal eminences enlarged, the skull unsymmetrical, and the bridge of the nose depressed. The teeth are often poorly developed and present the characteristics of Hutchinson's teeth—that is, the permanent upper incisors are short and narrow and peg-shaped, and at the cutting borders there are shallow or deep notches in which the dentine is exposed. The other teeth also are usually deformed, but the changes in the upper central incisors are the most characteristic. Hutchinson's teeth, while suggestive of syphilis, are not pathognomonic, and are found in other nutritional disturbances. Among later disturbances seen in congenital syphilis are keratitis, less commonly iritis, ear affections, periosteitis, rarely joint lesions, and at times visceral gummata.

General glandular involvement is not as characteristic of congenital syphilis as of acquired, although in abundant eruptions the contiguous glands are usually enlarged.

The diagnosis of congenital syphilis is rarely difficult. The general cachexia, the old-man appearance, the snuffles, the hoarse cry, the excoriations of the lips and the fissures at the angles of the mouth, the fissures and condylomata about the anus, and the peculiar eruptions make up a clinical picture that is as characteristic as it is pitiful.

The prognosis of congenital syphilis is bad. The majority of the children die—well over sixty per cent.

ETIOLOGY

Syphilis is an infectious disease undoubtedly due to a specific organism, and is transmitted only by inoculation or by hereditary transmission.

During the primary and secondary stages of syphilis—that is, during its existence as an active constitutional disease—the blood and the lymph and the organized material and the secretions of the lesions contain the virus of the disease and infection may occur from any of these. The blood and lymph are, perhaps, the least infectious and lose that character first. If unmixed with the infection-bearing materials the normal secretions—sweat, tears, saliva, milk, urine, and semen—

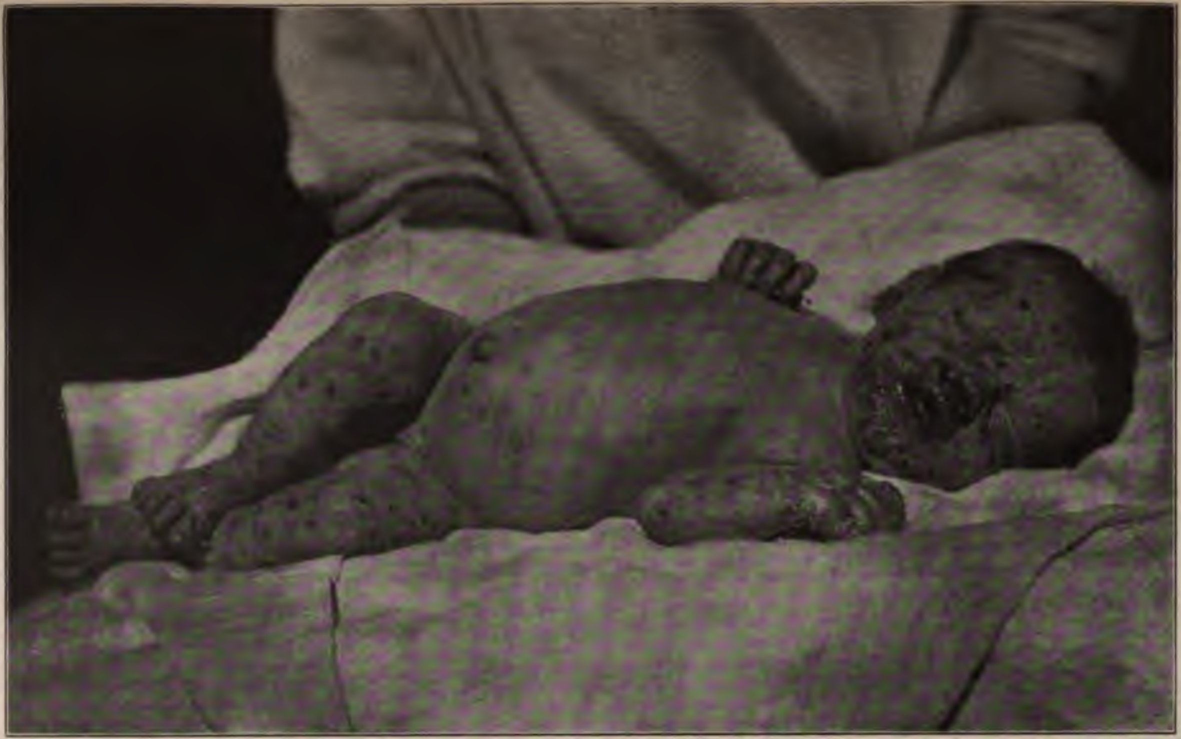

Fig. 168. — Congenital Syphilis. (Author's collection.)

do not contain the virus. Recent investigations throw doubt upon the accuracy of this statement as regards the semen.

The poison of syphilis can maintain its virulence for a short time outside of the body, so that infection may be not only direct but indirect, through contact with substances of any sort upon which the poison has found lodgment. The disease is in the great majority of cases transmitted by direct infection.

The lesions which are practically most dangerous as sources of infection are chancres, mucous patches, and condylomata. In the majority of cases syphilis is acquired from the chancre. Perhaps next most frequently it is acquired from vaginal mucous patches. Lesions about the mouth also are very dangerous, and they have a special importance because they are so frequently the cause of innocent syphilis.

For infection with syphilis a break in the epidermis is necessary, but this may be of microscopic size. It is maintained as a possibility that infection may take place through the delicate unbroken mucous membrane and through the cutaneous follicles by the poison finding lodgment in them, but it is the constant experience of those who have to do with syphilis that there is practically no danger of infection with syphilis so long as the skin is absolutely intact.

It is hardly necessary to consider in detail the innumerable means by which the virus of syphilis may be transferred, by either direct or indirect contagion. Anything which serves as the bearer of the syphilitic poison to an abrasion of the human skin may be the means of infection. Syphilitic infection results in a large majority of cases from inoculation during coitus. It is by no means rare, however, for syphilis to be of nonvenereal origin. According to Bulkley's statistics, in about five per cent of the cases the primary lesion is extragenital.

The virulence of the syphilitic poison and the danger of the transmission of syphilis is greatest in the early active stage of the disease. There is no appreciable diminution in its infectiousness during the continuance of the florid active manifestations of the secondary period, but as the evidences of active constitutional infection diminish and secondary lesions disappear the infectious capacity of the disease gradually diminishes and finally vanishes. The time of the disappearance of infectiousness in syphilis cannot be stated in months. In thoroughly treated cases in which the manifestations of the secondary period are moderate and quick to disappear, infectiousness may cease by the end of the first year, and its danger is certainly greatly diminished by that time. It is likely to disappear by the end of the second year; after three years contagion is almost unknown, and, according to Hutchinson, after five years never occurs. The late tertiary lesions are not, as a rule, infectious, but there are instances of the transmission of the disease from late tertiary lesions which were numerous and virulent.

The finding of spirochætes in gummata and inoculation experiments on apes, using material from gummata, have demonstrated that late lesions are infectious. In these lesions the active border, not the pus, contains the virus, so that the danger from these lesions is vastly less than from lesions occurring during the time when the disease is generalized and all lesions are teaming with spirochætes.

Transmission of Hereditary Syphilis.—Congenital syphilis may be transmitted from the father alone, from the mother alone, or from both. Maternal transmission is more certain and more potent for harm than paternal, and the danger of its occurrence persists longer, but is less frequently seen than paternal transmission.

Hereditary syphilis may be acquired from a syphilitic father by direct inoculation of the ovum—seminal transmission—without the production of manifest evidence of syphilis in the mother. This is more frequent than maternal transmission. It may be acquired from the mother as a result either of active syphilis existing at the time of infection, or of infection at the time of conception, or of infection subsequent to conception. Congenital syphilis acquired from post-conceptional syphilis is less impressed upon the ovum than when infection exists from the time of conception, and the disease is likely to be less intense. If infection occurs as late as the seventh month of pregnancy the child is likely to escape congenital infection, although there are cases of congenital syphilis from mothers who developed the initial lesion as late as the eighth month. If both father and mother are infected at the time of conception the danger of transmitting congenital syphilis and the severity of its manifestations, if it is transmitted, are greatly increased.

The capacity to transmit syphilis to the offspring persists perhaps somewhat longer than the danger of its transmission by contagion, but it diminishes rapidly upon the disappearance of the active evidences of constitutional infection. After three years in well-treated cases syphilitics usually beget healthy children, and after four years the chance of a syphilitic offspring is excessively remote.

The possibility of the transmission of syphilis through a congenital syphilitic to the third generation is an unsettled question. Burgsdorf, accepting interstitial keratitis as critical evidence, has reported such a transmission in a child five years old. If transmission to the third generation ever takes place, it is the rarest of rare occurrences.

SUSCEPTIBILITY.—Perhaps no person who has not had hereditary or acquired syphilis is immune to syphilitic inoculation, but there is reason to believe that some individuals are less susceptible than others. Differences in susceptibility, however, are shown chiefly by the variation in intensity in the disease after it is acquired. The greatest susceptibility seen in those persons in whom the disease appears as malignant hilis. Actual immunity to syphilis is found:

(1) In those who have had acquired syphilis.

(2) In those who have inherited syphilis (Profeta's law).

(3) In mothers who have borne syphilitic children, even though ey have themselves shown no other evidences of the disease (Colle's w).

To all of these statements there are rare exceptions, but they are ceedingly rare. In rare instances the immunity resulting from a pre-ious attack of the disease wears away after many years, and there are thentic cases of reinfection with syphilis years after the first infection. here is, however, no authentic case of the reinoculation of syphilis in n individual in the active secondary stage of the disease, or within five ears of the initial lesion.

As already indicated, probably the most potent factor in determining e severity of syphilis is the resistance of the individual to the poison. he resistance may be naturally high or low, and this results in corre-ondingly mild or severe manifestations of the disease when it is ac-ired. In old age and in childhood, in patients who are not vigorous, ho have some constitutional dyscrasia, who are reduced by disease, by hygienic living, or excesses, especially alcoholism, the manifestations syphilis are likely to be more severe. As syphilis is seen in the better ss of private patients it is, as a rule, of very moderate severity, and the ere forms of the disease are seen almost exclusively in dispensary and pital practice.

The most important factor in the etiology of tertiary syphilis is lack thorough treatment. Leaving this out of consideration, tertiary le-ns are most likely to occur in cases which have been severe during the ive period of the disease.

There seems to be a general amelioration of syphilis in recent times. e severe manifestations of the disease recorded by the older writers we now rarely or not at all. The explanation of this is a matter of sur-se; perhaps some light is thrown upon it by the well-known fact that hilis, like other infectious diseases, occurs in a much more virulent m in peoples among whom it has not previously existed.

PARASITOLOGY

There have been few searches more zealous than that which has been ade for the organism of syphilis. Lassar, in the discussion of Schaudinn d Hoffmann's paper announcing the discovery of a new organism in philis, stated that 125 causes of syphilis had been announced in the st twenty-five years. Of these, few have been entitled to serious consideration. The one which has received most attention is Lustgarten's bacillus which has been definitely shown to be, usually, at least, saprophytic and certainly not specific. In recent years Siegel has insistently urged the pathogenic character of certain bodies named by him cytoryctes luis, which he has found in blood and regards as the organisms of syphilis. Siegel's organisms are generally accepted to be blood platelets, cell granulations and cell degenerations, and are found in other conditions. They will remain of historical interest, however, because in a critical examination of them undertaken by Schaudinn and Hoffmann, as a committee, to determine their relationship to syphilis, the spirochæta pallida was discovered. This discovery was announced by Schaudinn and Hoffmann in May, 1905. Eighteen months previous, in December, 1903, Metchnikoff had reported the discovery that syphilis could be inoculated in anthropoid apes. These two facts mark an epoch in the history of syphilis. Metchnikoff's discovery opened up the field of experimental syphilis, and with Schaudinn and Hoffmann's discovery has made it possible to study experimentally all of the facts of syphilis, concerning which our previous knowledge was based solely upon clinical observation. In the last five years a vast amount of work upon this subject has been done by many investigators, notable among whom are Neisser, Hoffmann, and Finger; Schaudinn's death in 1906 removed the

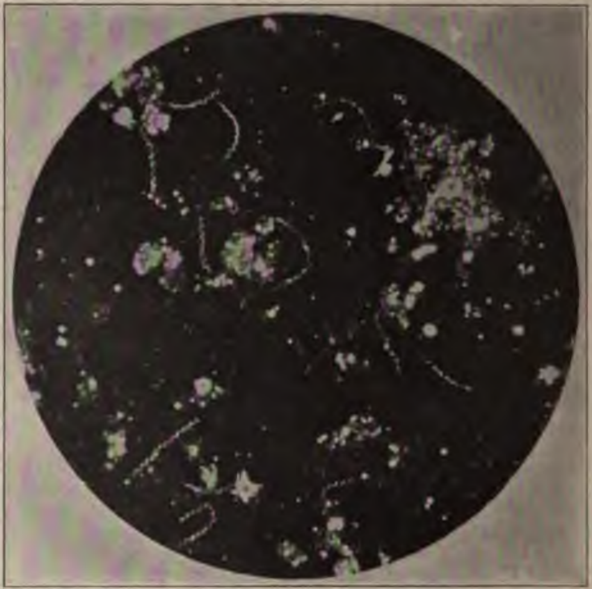

FIG. 169.—SPIROCHÆTÆ PALLIDÆ SHOWN BY DARK-GROUND ILLUMINATION. × 1000. (Published through the courtesy of Professor Hoffmann.)

one who was best fitted by training for the study of the parasitology and is an irreparable loss.

Many spirilla have been found in syphilitic lesions previous to Schaudinn and Hoffmann's discovery. Schaudinn and Hoffmann, however, found two new spirochætes in the genitals. One, called by Schaudinn the spirochæta refringens, is not confined to syphilitic lesions and is readily distinguished by its morphological characteristics from spirochæta pallida; the other, which was named the spirochæta pallida, was found in all lesions of syphilis which they had examined and in no other affection. It occurred not only on the surface of syphilitic lesions, but in the depths of tissues and in syphilitic glands. Workers in great numbers all over the world have abundantly confirmed the findings of Schaudinn and Hoffmann, and the grounds are very strong for believing that the spirochæta pallida is the organism of syphilis.

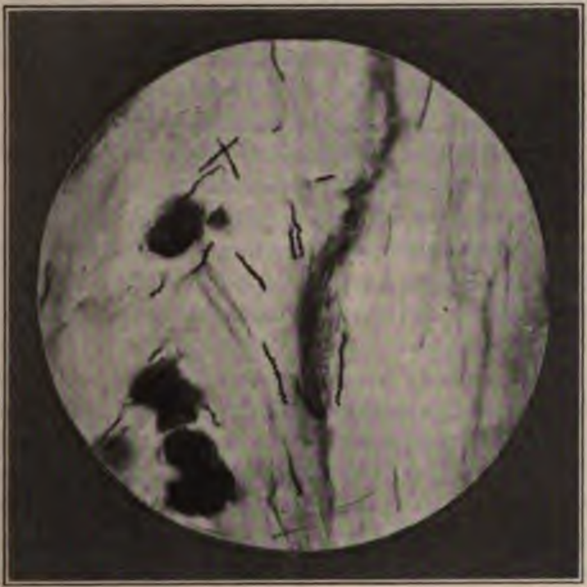

FIG. 170.—SPIROCHÆTÆ PALLIDÆ IN THE CORNEA OF A RABBIT.
(Published through the courtesy of Professor Hoffmann.)

The spirochæta pallida averages in length from six to fifteen microns. Occasionally organisms are twice as long. The diameter of the spirochæta is very small, about one fourth of a micron. Its undulations are characteristic and peculiar in that they remain the same during rest as well as motion. Schaudinn, in his work published by Hartmann and Prowazek after Schaudinn's death, describes the organisms as follows:[1]

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[1] Quoted from Metchnikoff.

"In comparative research we found at the very start that the spirochæta pallida, which is only found in syphilitic lesions, varies extremely little in appearance; quite unlike the other known varieties of spirochætes. As soon as one has had experience it is extremely easy to distinguish it from other forms when it is in the living state. The delicate shape and slight refractive power of spirochæta pallida, together with its typical spiral form with very numerous undulations (ten to twenty-six) which are perfectly regular, well marked and closely resembling each other, make it very difficult to mistake it for any other object whatever. The following fact is of particular importance and is to be observed when examining the living organism. It is that the typical spiral form is to be seen not only when it is actively moving, but also when it is at rest, whilst all other analogous spirochætes which present marked undulations show these only when they are moving vigorously, and when at rest have long flattened-out undulations and, indeed, are very nearly straight. . . . There is only one spirillum, that of the mouth or spirochæta dentium, which has a similar spiral arrangement, but this variety can be distinguished by other characteristics from the organism of syphilis."

The facts concerning spirochæta pallida and its relationship to syphilis which have been established by the investigations resulting from Schaudinn and Hoffmann's discovery, put as briefly as possible, are as follows: There has been unanimity in the confirmation of the fact that the spirochæta pallida is demonstrable in all syphilitic lesions, and there is little dissent from the view that it is a hitherto unknown organism with distinguishing morphological characteristics, and that it has not been found except in syphilis. The organism has been found in all of the cutaneous lesions of secondary syphilis, in syphilitic glands, in circulating blood; in acquired syphilis, in the heart, the blood vessels, the liver, the spleen, the cerebro-spinal fluid, the testis, the ovary and in young ova, in the placenta and umbilical cord, and in congenital syphilis in practically all organs and tissues. Inoculation experiments have shown that after the introduction of syphilitic material containing spirochætes, there is first a growth of the organisms which after a time results in sufficient irritation to produce a local proliferation of syphilitic cells. From this local focus the organisms are disseminated first by the lymph channels and then by the blood vessels. The organisms do not remain long in the blood, as indicated by the difficulty of demonstrating them in the blood, but invade the tissues, showing a special predilection for the skin, lymphatic tissue, blood vessels, glandular organs, and bone marrow. The organisms show a marked tendency to persist at the point where they have found opportunity to proliferate in sufficient numbers to produce a syphilitic reaction. They can be found as long as the least degree of infiltration persists and at the sites of eruptions even after the lesions themselves have disappeared. They are found, but with difficulty, in the late lesions of the disease. Corbus and Harris found them in a mucous patch in which the infection was of eight and one half years' duration. Neilson found them in a papular erosive lesion of the mouth nine years after infection. It has been demonstrated that they occur in gummata regardless of the period of the disease. Their numbers vary with the acuteness of the lesions, the more active the lesions the more abundant the spirochætæ. In late gummata they are not found in the degenerating mass or in the pus, but only in the active border. As a rule, they are found in the intercellular spaces of the tissues, sometimes they are intracellular. In active lesions they are most numerous in the center of the lesions, diminishing toward the border, but extending even beyond the inflammatory area. In congenital syphilis a very remarkable fact is the great abundance of the spirochætæ in organs in which there may be no evidence of inflammatory changes. The local use of mercurials in open lesions causes rapid decrease in the number of spirochætæ present, while they remain abundant in contiguous glands. The internal use of mercury makes their demonstration in skin lesions much more difficult.

In order to study the living organisms of syphilis, the dark ground illuminator has been introduced. This has made the examination for spirochætæ a simple procedure, and in addition to its simplicity the method has the advantage that it brings out strongly the morphological characteristics of the organism so that it can be readily differentiated from other spirochætæ. This method furnishes an easily and readily applied means for diagnosticating syphilitic lesions by demonstrating the presence of the spirochæta pallida. It is particularly useful in confirming the diagnosis during the first manifestations of the disease. The method of examination is as follows: The suspected lesion is first cleaned with alcohol and water and dried. Then, if it is a weeping lesion, serum from the surface is collected in a sterile capillary pipette; if there is no free weeping, the cleansed dry surface is irritated with a cotton swab until serum exudes. In a lesion covered by epidermis the surface should first be abraded and then cupped with a small cupping glass, or, better, a vesicant is applied for several hours and the serum from the blister is collected in a pipette. In obtaining material from a gland the needle is inserted into the mass of the gland and serum collected in a syringe. In all of these procedures the aim is to get the serum as free as possible from red blood cells. After the serum is collected in a pipette it is blown out (with a rubber bulb) upon the slide of the dark ground illuminator and examined by transverse illumination. The characteristics of the spirochætæ pallidæ, as seen in the dark ground illuminator, are as follows (Harris): (1) They vary in length from seven to twenty-one microns, about one to three times the diameter of a red blood cell. Longer forms sometimes seen are probably composed of two or more individuals; (2) The body is an extremely slender thread regularly wound in corkscrew form, the spirals being very acute and usually numbering from eight to fourteen in an organism. In fresh preparations the spirals are absolutely regular; in the older organisms their form changes somewhat, usually by the irregularity or obliteration of the windings in the middle. The ends of the spirochæta pallida are sharp and terminate on the periphery of the spiral and not in the center as in other spirochætæ; (3) Fresh organisms have three motions: First, a rapid rotation on the long axis, in either direction, which is not necessarily accompanied by change of position; this motion decreases with the age of the specimens. Second, a progressive motion which is less rapid than in other common forms of spirochætæ. Third, a lashing and twisting motion which increases as the specimens grow older. Occasionally the organisms will be bent almost into circles or hoops.

Recently the cultivation of the spirochæta pallida on artificial media has been announced. Schereschewsky has cultivated the spirochæta in pure horse serum, using material obtained from a syphilitic papule or condyloma. In his cultures there was a mixture with other bacteria. Mühlens has, however, obtained pure cultures of the spirochæta pallida from a syphilitic gland. These in the fourth generation were identical morphologically in living and stained specimens with the original organism. Schereschewsky reports failures thus far in animal inoculations; Mühlens has not yet reported upon his experiments.

Experimental Syphilis

The knowledge derived from the experimental study of syphilis in animals following Metchnikoff's discovery and in the light of the recognition of the spirochæta pallida may be summarized as briefly as possible as follows: First, it is interesting to note that Klebs, in 1879, claimed that syphilis could be inoculated into apes, and Hensell, two years later, announced that syphilis of the iris and cornea could be produced in rabbits. Metchnikoff's early observations were confined to anthropoid apes. Later experiments soon showed that, while higher apes were more susceptible, monkeys in general are susceptible to syphilis. In the higher apes inoculation anywhere in the skin is almost invariably successful. In the lower apes inoculation usually fails except on the eyelids, eyebrows, and genitals. In the higher apes the disease is demonstrated by a disseminated eruption, while in the lower apes the disease may only appear as a localized eruption around the site of the disease. Hoffmann and Lohe, however, in nine lower apes inoculated, had disseminated eruptions in three cases. Experiments show that the organisms take root much more readily in some tissues than in others. The skin and the testes are the most susceptible sites of inoculation. The eyelids, eyebrows, and genitals are the most susceptible sites in the skin, and inoculations superficially in the skin are more liable to be successful than those made deep in the skin. Subcutaneous, intravenous, and intraperitoneal inoculations have uniformly failed.

Barterelli, and, after him, Greef, Schucht, Hoffmann and others have confirmed Hensell's original observation that the disease can be inoculated into rabbits' eyes with the production of a keratitis, sometimes of an iritis. Successful inoculations have also been made in dogs' and guinea pigs' eyes. From an infected cornea in a rabbit Hoffmann has produced in a monkey an initial lesion from which he recovered the spirochætæ.

The primary incubation period in animals averages about thirty days. It is somewhat shorter in apes than in monkeys and rabbits, but has been as long as fifty days. There results an initial lesion corresponding clinically and histologically to that occurring in man. In apes this is followed by a general dissemination of the disease with papules and erosive lesions of the skin and mucous membranes. True tertiary lesions have not yet certainly been observed.

Inoculations have been made successfully from mucous patches, papules, chancres, and contiguous glands; from the mucous in syphilitic coryza, from spinal fluid (once in three times), from the blood—not from blood serum—from seminal fluid; of internal tissues in acquired syphilis, only from the spleen, the bone marrow, lymphatic glands and testes, but in congenital syphilis positive results have been obtained by inoculations from the lung, suprarenal capsule, kidney, liver, ovary, testis, bone marrow, and heart blood. From gummata, inoculations have been successful in about one third of the cases, but only in these cases from the active growing border. Broken-down gummata and pus from gummata have not given positive results.

Whatever the source of the inoculation material the primary lesion is the same. There is no evidence of attenuation of the virus from long duration of the disease in any subject. Late lesions or other tissues, like the blood, which contain spirochætæ only sparsely require a relatively large quantity of inoculating material in order to get a successful inoculation, but the disease produced from such inoculations does not differ in intensity from that produced by inoculations from early lesions.

Finger and Landsteiner have carried the infection without attenuation through forty-five different monkeys. Metchnikoff has attempted to produce an attenuated virus by passing the disease from the chimpanzee through lower apes in a series of twenty-two individuals, and he believes that he has gotten very marked attenuation. Others have not had the same results, and Neisser has even seen an apparent increase in the virulence in passing through monkeys. There is no evidence of any variation in virulence whether the source of the virus is animal or human, internal or external, early or late, hereditary or acquired. The experiments, therefore, are unfavorable to the hope of the production of an artificial immunity. Indeed, animal experiments indicate that syphilis does not produce a real immunity, but that the immunity only lasts so long as the disease exists. The experiments so far show that, in the case of apes which cannot be inoculated with syphilis, their tissues will produce the disease in other animals by inoculation.

The substance of experiments regarding the rapidity of the dissemination of the disease indicates that, as a rule, in about a day the virus penetrates beyond the possibility of local interference. Excision of the point of inoculation six hours and occasionally later may prevent the development of the disease. Excisions, eight, fifteen, and twenty-four hours after inoculation by Neisser have not prevented its development. In Neisser's experience rubbing the site of the inoculation with thirty-per-cent calomel ointment at various intervals up to twenty hours usually prevents the development of chancre, but rubbing with mercurial ointment ten minutes after inoculation failed twice. Metchnikoff and Roux are very firm in their belief that an ointment of one part calomel and three parts lanolin rubbed into the site of inoculation within from one to eighteen hours after inoculation—the sooner the better—prevents the development of the disease. According to their experiments on monkeys, the measure is not effective twenty hours after inoculation. Constitutional mercurial treatment begun simultaneously with inoculation has failed to prevent the development of the primary lesion.

Experiments indicate that the disease has become general when the primary lesion appears. Spirochætes have been demonstrated in the internal organs within ten days after inoculation, and infectious material has been obtained from the spleen on the fifth day, from the blood on the fourteenth day, and from the bone marrow on the fifteenth day.

Reinfection.—Inoculation during the primary period may be followed by a typical lesion. After the secondary period develops, inoculation fails.

Persistent attempts to preserve active the virus in syphilitic material after removal from the living body have been uniformly unsuccessful. Virulence disappears from infectious material at the end of the sixth or seventh hour after removal from the body. Inoculations made directly from the cadaver were always negative after an interval of eighteen hours. Metchnikoff and Roux determined that the virus will not pass through a Berkfeld filler and is destroyed by heating to 51° C. for an hour.

A very few cases are recorded of infection from syphilitic cadavers, in one case twenty-four hours after death. (Gross, *Jour. A. M. A.*, February 9, 1907, p. 538.)

HISTOPATHOLOGY

The pathological anatomy of the papule is typical of the cutaneous lesions of syphilis, all of which, except the roseola, may be considered simply as variants in extent, severity, or secondary accidents. The papule is a dense, at first perivascular, granulomatous infiltration of the papillary layer of the corium, consisting of plasma cells, with some mast cells. The collagen bundles are not destroyed, but are increased in size and number. New-formed capillaries traverse the infiltration, there is escape of the red blood cells, leading to the raw-ham color, and there are proliferative changes in the vessels. The plasma cells are frequently arranged in layers between the collagenous bundles, and occasionally form giant cells. The cells of syphilitic infiltrations undergo degeneration leading to absorption or ulceration, but do not become organized into connective tissue. The overlying epithelium is secondarily affected, showing proliferation, œdema, or necrosis.

In the initial sclerosis the infiltration is very dense, and the cells are thickly packed between hypertrophic and hyperplastic collagenous bundles. Over the center the epithelium shows parakeratosis and atrophy leading to desquamation and excoriation; peripherally there is proliferation, with processes growing downward into the less dense portion of the infiltration.

In the larger lesions the granuloma extends through the whole extent of the corium, and even into the subcutaneous tissue. Frequently the collagenous bundles form a peripheral capsule, within which, as a result of pressure from the dense infiltration, the cells break down, the collagen undergoes colloid degeneration, and a caseous mass results which is thrown off by ulceration. The central pressure may lead to ulceration or absorption followed by cicatricial formation, even while the process is extending peripherally.

The cells of the syphilitic granulomata are very persistent, often remaining for three to six months or longer after the lesions have disappeared clinically.

The pathology of the roseola is that of any other toxic erythema.

DIAGNOSIS

The establishment of a positive diagnosis is, in syphilis more than in most other diseases, a matter of the greatest practical importance. It involves not only the question of treatment, but other matters of vital interest to the patient. If a patient has syphilis he should submit to a long course of treatment; his social relations must largely be governed by the fact that for a year or more he is a possible source of contagion; he must refrain from marrying, or, if married, he must take the greatest precautions to avoid infecting his family, and must not have children until the active disease has passed. Further, he has always the remote danger of the appearance of gummata in any of the tissues of the body; and if, in later life, he should be the victim of a neoplasm, the fact of his having had syphilis would be of the utmost importance in deciding upon its character and treatment. Further, he is the possibl victim in later life of one of the parasyphilitic affections. These possi bilities, all of them of great practical importance, render it imperativ to establish positively, if possible, in a suspicious case, whether it is o is not syphilis.

In making a diagnosis of syphilis consideration should be given t all ascertainable facts. In active secondary syphilis the facts ascertai able without the coöperation of the patient are sufficient to render th diagnosis, as a rule, easy. In tertiary syphilis the diagnosis must ofte be made, without history, upon the characteristics of the cutaneous l sions or upon a small group of symptoms in more deep-seated lesion Fortunately, in the cutaneous lesions of tertiary syphilis the objectiv symptoms are, as a rule, so characteristic as to make the diagnosis cle to the expert; and further, where there is doubt, it can in most cases l cleared up by a few weeks of antisyphilitic treatment.

The important features in the diagnosis of syphilis are: the defini evolution of the disease through an incubation period, a primary perio and into a secondary period; the indolent, painless character of the i itial lesion and its persistence for two weeks or longer; the development i definite sequence after the initial lesion of general glandular enlargemen and the appearance in definite sequence of the evidences of a low grade constitutional infection and of lesions of the skin and mucous membrane When the patient is seen in the active secondary stage, all of these fac can usually be determined whether one have the coöperation of the p tient or not. It may be impossible to obtain a satisfactory history of th primary incubation, of the initial lesion, and of the secondary incubatio period, but the induration remaining after the primary lesion can us ally be found, and, given this in conjunction with the general adeno athy, mucous patches, and cutaneous lesions, with or without moi lesions about the genitals and anus, the diagnosis offers no difficulty. Th clinical situation is characteristic and definite and, with ordinary ca in ascertaining the facts, should not be confusing. Mistakes are muc more liable to occur if too much reliance is placed upon one feature, an the entire symptom-complex is not taken into consideration. The expe can usually make a definite diagnosis upon the cutaneous lesions alone, but he is relieved if he can fortify his decision by the finding of at least general adenopathy and mucous patches.

In tertiary syphilis one is often thrown completely upon the objective characteristics of the cutaneous lesions for his decision. Frequently the situation is such that it is impossible to go into the question of history, and when this is not so it is the common experience of those who have to do with syphilis that in a surprisingly large number of cases no reliance can be placed upon a negative history. The early manifestations may have been so light as to have escaped the patient's notice, or, more frequently, the patient does not give a truthful report. If a positive history of syphilis is obtainable it is of some value in determining the character of a probable tertiary lesion; but a negative history should in many cases have practically no influence in one's decision concerning a suspicious lesion. Fortunately tertiary lesions in the skin are usually so characteristic that they allow definite diagnosis. The characteristic features have been taken up in considering the lesions. When in doubt, light may be thrown upon the character of the lesions by microscopic examination, and the character of a tertiary lesion can usually be absolutely determined by two weeks of antisyphilitic treatment.

Complement-Fixation Test in Syphilis.—The principle of the complement-fixation method of Bordet and Gengou, which was applied by them first to a test for typhoid fever, has been applied by Wassermann, Neisser, and Bruck to a complement-fixation test for the diagnosis of syphilis. The principles involved in the method and the technique of the Wassermann test, and of the various modifications of it which have been suggested, are complicated, and the statement of the technique in sufficient detail for carrying out the test in practice requires an amount of space which can only be given in extensive articles upon the subject. For the principles and technique of the test, therefore, reference must be made to the articles given in the bibliography below or to the voluminous literature upon the subject.

The Wassermann serum-complement reaction has been given a very wide and critical examination during the last four years. An enormous amount of investigation has confirmed its value in the diagnosis of syphilis. Upon this point there is practical unanimity of opinion. The test possesses the first essential of a diagnostic test in that it is not misleading. It does not give a positive reaction in the absence of syphilis, except in the case of a few diseases which are readily distinguished from syphilis. Positive reaction has been obtained in leprosy, scarlet fever, acute diabetes and noma, which are readily distinguishable from syphilis. Fortunately tuberculosis and cancer have been found not to give positive reaction. The findings of many investigators show that the reaction is positive in about eighty-five per cent of the various types of syphilis. The reaction does not develop, as a rule, until the initial lesion has been present about a week. From that time it persists as long as syphilis remains active. As would be expected from the factors of uncertainty in any series of cases, the findings of different investigators show some variations in the different stages of the disease, but there is fairly close unanimity in the findings of various workers, showing a positive reaction in eighty to ninety per cent of cases in all stages of syphilis. This applies, of course, not only to cutaneous and demonstrable lesions of syphilis, but to visceral lesions and latent syphilis. It is an interesting fact that about the same findings hold for tabes dorsalis and paresis. In congenital syphilis the reaction is strong.

The Wassermann reaction is very greatly influenced by mercurial treatment. This effect of mercury upon the reaction is very much more marked in active secondary syphilis than in late syphilis. Under vigorous, persistent treatment during the secondary period of syphilis the reaction tends to disappear. In late syphilis this disappearance after vigorous treatment is less frequent. In contrast to the disappearance of the reaction in vigorously treated cases, Bruck and Stern in experiments upon monkeys found that in untreated cases the reaction did not tend to disappear within the two years during which the monkeys were under observation. It is evident that the findings from the Wassermann reaction gives valuable information as to the continuance of mercurial treatment. There is strong reason for holding that during the first years of the existence of syphilis treatment should be continued as long as the reaction remains positive. The reverse of this does not hold, and the findings do not indicate that treatment should be discontinued with the disappearance of a positive reaction during what is clinically the active period of the disease. On the contrary, they indicate that the eradication of the disease, so far as it is shown by the complement-fixation test, is the more likely when vigorous treatment is persisted in throughout the years of the active period of syphilis.

The serum-complement reaction in the diagnosis of syphilis is undoubtedly of great value, but it is open to error. When properly performed, with scrupulous attention to detail, particularly to the control tests, a positive reaction is of great diagnostic value. It must be remembered, however, that a negative reaction does not absolutely exclude syphilis, and due attention must be paid to this fact when a negative reaction is obtained. To properly carry out the test a careful technique and experience are necessary, and there is no doubt that the reaction is liable to great abuse at the hands of inexperienced workers. The greatest danger which lies in the reaction is that it shall be accepted as a finality without regard to the clinical findings. The only safe way of using the test is to remember that it is but one fact in the diagnosis of syphilis and that it must not be given sole consideration in forming an opinion concerning any given lesion to the exclusion of equally definite clinical facts.

PROGNOSIS

The prognosis of syphilis, as a rule, is good. In patients of reasonable resistence who are properly treated, the active secondary manifestations of the disease rarely pass beyond the macular eruption, glandular enlargement, mucous patches in the mouth, and slight cachexia. After the first few months the patient is not aware of the presence of the disease, except perhaps for slight glandular enlargement and the occasional occurrence of a mucous patch. In unusual cases which do not do so well, the manifestations vary in intensity, but it is excessively rare to see cases, in otherwise healthy persons, which are in themselves troublesome. The difficulties arise from the social complications.

In persons who are otherwise diseased, syphilis is more difficult of management and may prove a much graver affection. The complication of syphilis with tuberculous is regarded as a particularly bad symbiosis, chiefly, however, through the unfavorable effect of the syphilitic cachexia upon the tuberculosis. In individuals who are reduced by dissipation, excesses, and unfavorable conditions of living, syphilis manifests itself much more severely, but even in these individuals the difficulty is chiefly that they do not have thorough and regular treatment. It is, therefore, in pauper practice, especially in public hospitals, that we see the severe manifestations of syphilis. The chief exceptions to this statement are the destructive manifestations of tertiary syphilis which are not infrequently seen in private practice because of failure to diagnosticate the disease.

The weak point in the prognosis of syphilis is the fact that we cannot positively say that the patient will never have a tertiary manifestation. The possibility of the occurrence of tertiary lesions is greatly reduced by efficient treatment during active syphilis, but the possibility of tertiary lesions in later life can never be absolutely excluded. The same applies to the so-called parasyphilitic affections. Syphilis would be, except for its social complications, a comparatively unimportant disease when thoroughly treated were it not for the fact that the syphilitic in after-life is always confronted with the possibility, remote though it be, of the development of a gummatous lesion in the nervous system, or of locomotor ataxia, general paresis, arterial sclerosis, or leukoplakia.

As to the question of the marriage of syphilitics, we have seen that syphilis as an infectious disease is in most cases well by the end of two to two and a half years. It is certainly not safe, however, for the syphilitic to marry until three years after the initial sclerosis, and at least a year after the last manifestations of the disease, and to be absolutely safe he should wait four years. After four years, if there has been no manifestation of the disease since the end of the second year, he can marry with a practically safe assurance of having healthy progeny.

Prevention of Syphilis

Attention has been called to the prevention of experimental syphilis by inunction of 33 per cent calomel within eighteen hours after infection. In view of this possibility, whenever a possible infection comes under observation within twenty hours, vigorous inunctions of thirty-three-per-cent calomel ointment preferably made with lanolin should be used. The ointment should be rubbed in for five minutes repeatedly at intervals of a few hours, and during the intervals should be applied under a dressing. Metchnikoff's ointment is calomel 33 gms., lanolin 67 gms., vaseline 10 gms.

Treatment

Preliminary Treatment.—Upon the appearance of a lesion suggestive of chancre it should be taken care of according to surgical principles after the methods to be considered under local treatment. At the same time measures can be taken to prepare the patient for the onset of the disease, in case it proves to be syphilis. Carious teeth should be filled or taken out, tartar removed, and the patient impressed with the importance of the scrupulous care of the mouth. Frequent brushing of the teeth and gums should begin at this time, along with the use of a dentifrice and an astringent, mild antiseptic mouth wash, such as liquor antisepticus or solution of chlorate of potash or of boric acid. If there are disturbances of the gastrointestinal tract, an attempt should be made to remedy them, and in general every effort possible should be made to have the patient in as vigorous condition as possible. The skin requires no special attention; hot bathing should be avoided, as it is likely to exaggerate a specific eruption, but bathing for cleanliness should be looked after, and there is no objection to the cool tonic bath if the patient is in the habit of taking it. One cutaneous condition which frequently complicates syphilides may warrant looking after at this time, and that is seborrhœic dermatitis, or seborrhœa of the face. This exaggerates syphilides of the face, and as it is very frequently present it should be attended to at this time.

It is not probable that more than this can be done to good advantage during the second incubation period.

For a long time efforts have been made to abort syphilis by excision

EHRLICH'S "606"

At the present time intense professional and public interest is aroused over a new remedy for syphilis introduced by Ehrlich and Hata. With this agent Ehrlich has made no less an attempt than to destroy entirely the organisms of syphilis in the human body by one injection of a massive dose of arsenic furnished in this synthetic compound. The remedy, which Ehrlich has called "606," is the result of a definite effort made by him to produce a synthetic compound which, to use Ehrlich's term, would be highly "parisitropic," for such organisms as spirocheta pallida, and at the same time be, to use Ehrlich's term again, devoid of "organotropic" activities, and so, relatively harmless.

The following comments upon the chemistry of "606" are from the *Journal of the American Medical Association*:

"Its structural formula is: $NH_2.OH.C_6H_3.As{:}As.C_6H_3.OH.NH_2$.

"To understand the chemical properties of this body, it should be noted that the arsenic is in the unstable trivalent form and not in the stable pentavalent form, as in sodium cacodylate, atoxyl, or arsacetin. Arsen-phenol-amin (or "606") is very unstable and is put on the market in the form of its hydrochlorid, which, owing to its acid character, however, causes injections of it to be very painful. For this reason the hydrochlorid is treated with an amount of alkali exactly sufficient to combine with the hydrochloric acid of the salt and to liberate the base arsen-phenol-amin which is insoluble and which is injected in the form of a suspension in water."

The recent technique recommended by Wechselmann is as follows:

The dose, 0.3 to 0.6 gm., is dissolved, best in a small sterile mortar, in 1 or 2 c.c. of normal sodium hydrate solution. Acetic acid is added drop by drop until the base precipitates in a fine yellowish powder. After the precipitation, it is important to rub the mixture thoroughly in order to divide evenly the particles and make a homogeneous paste. One or two drops of sterile water are added to the paste and then 0.1 normal sodium-hydrate solution, or 1 per cent acetic-acid solution, as necessary according to the reaction, is added until the solution is exactly neutral to litmus paper; the more exactly neutral the reaction of the mixture, the less painful the injection. Of course the technique must keep the agent sterile, and aseptic precautions must be used in the injections.

Mode of Administration.—The dose of the agent which has thus far been used is 0.3 to 0.6 gm., the tendency being to increase the dose. As experiments have shown the toxic dose in animals to be not less than 0.015 gm. per kilo, it is thought that this dose may be subsequently much increased. The agent is stable only in the form of hydrochlorid, which is strongly acid when dissolved in water, and it is converted just before use into a mono- or by-sodium salt by the addition of sodium hydrate. The injections may be intramuscular, intravenous, or subcutaneous. The intramuscular injections are the most painful and are most likely to be accompanied by febrile reaction, perhaps as the result of irritation from sodium hydrate. They are given in the buttocks and are followed by painful induration. The pain disappears shortly, but the induration is likely to persist. Pulmonary embolism has occurred (Hoffmann), as after intramuscular injections of mercury. The intravenous injections are less painful and less likely to be followed by fever, but Fraenkel and Grouven have had a death from acute arsenic poisoning after intravenous injection. The subcutaneous injections are therefore being given preference, and the preferable area for these is in the back below the shoulder blades. The subcutaneous injections are accompanied by slight pain lasting a few minutes and occasionally by a moderate inflammatory swelling on the second or third day. Of the three fatal cases of which Ehrlich knows, one has followed each of these methods of administration. He suggests that possibly the preferable technique would be to give part of the dose first by an intravenous injection and follow it in forty-eight hours with an intramuscular injection.

Following Ehrlich, the warning is constantly given that the injections are contraindicated in the presence of pathological conditions in the fundus of the eyes, the circulatory system, and the kidneys. Patients should always have a preliminary physical examination.

After the injections the excretion of arsenic appears promptly in the kidneys. Fraenkel and Grouven investigated the excretion of arsenic and found the elimination in the urine to be 6 to 10 mg. daily during the first week and 6 to 8 mg. in the second week, with slight excretion after that. Alt, in cases of nervous syphilis, found the excretion continuing longer and still going on ten to twelve days after injections.

There is unanimity of opinion as to the prompt effect on syphilitic processes. Primary lesions and secondaries disappear rapidly and ulcerative processes yield even more strikingly. Similar rapid beneficial effect is observed in brain and other nervous lesions. The marked improvement in lesions is apparent within two or three days and complete healing of extensive ulcerative processes may occur within ten days. The results have been particularly striking in some cases in which mercury had failed, in resistant syphilides like those of the palms, and, as many observers testify, in malignant syphilis. Pick reports that in ten cases in which injections were given before the appearance of secondaries no secondaries developed. But in Neisser's experience the remedy does not prevent the development of the disease, even when given in the presence of primary lesions before the Wassermann reaction is positive, although in apes he thinks it delays and modifies the primary lesions. Equally as striking as the resolution of lesions is the improvement which usually takes place in the general physical condition of the patients. There is disappearance of anæmia and cachexia, increase in weight, and a taking on of new vigor.

These favorable clinical results are even more rapid and definite than from the use of mercury, and have left no doubt of the very definite immediate action of the drug. The time is too short to properly estimate the frequency or the character of recurrence after injections, although such recurrences have been reported. Herxheimer in 200 patients has had only 1 recurrence, while Kromayer in 27 cases found the symptoms to persist after injection in 3 cases, and in 5 others has had recurring manifestations of syphilis.

Taege has observed the transformation into an apparently healthy babe of a congenital syphilitic child nine days old with bullous lesions after the mother's treatment by injection. He found that the drug did not pass into the mother's milk and attributes the effect on the child to antibodies produced in the mother by the sudden destruction of the spirochetes and the liberation of the endotoxins. Ehrlich has warned against treating syphilitic infants directly with "606" on account of the danger of serious intoxication from flooding the body with endotoxins, and the treatment of the infant by thus treating the mother is made a possibility.

After the injections there is usually within twenty-four to forty-eight hours complete disappearance of the pale spirochetes from chancres and condylomata, where they have before been very abundant. Herxheimer in 82 of 83 cases found no spirochetes forty-eight hours after injection. In the other case spirochetes were not affected, and Glück has had two similar cases, in which apparently the strain of spirochetes is insusceptible to the action of the drug. Iversen in 10 cases was unable to find spirochetes in aspirated fluid from inguinal nodes three to four days after injection.

The effect upon the Wassermann reaction is very much less definite and records are very conflicting. It is at least sure that the reaction is not reversed in a very considerable proportion of the cases. As competent observers as Isaac and Neisser report the persistence of a positive reaction in a large majority of the cases. Isaac in 27 patients found the Wassermann reaction still positive at the sixth week in all but one patient. Neisser has found a negative Wassermann in only about ten per cent of his cases, and this in cases which came under treatment soon after infection. He thinks that perhaps the failure to reverse the reaction is due to too small doses. Loeb has found only a very slight effect on the Wassermann reaction and Kromayer has gotten a negative in only twenty-five per cent of 27 cases. On the other hand, Schreiber and Hoppe report a negative Wassermann in 92.3 per cent of 57 cases. In their experience the earliest negative reaction was four days after injection and the latest seventy days after. In most cases they obtained it about fourteen days after. Iversen goes even further and reports a negative Wassermann within forty days after injection, in every one of 60 patients treated.

Certain by-effects are observed in the use of the agent. Local painfulness at the site of injection is usual, especially from intramuscular injections, and this is occasionally severe enough to cause great distress and require the use of morphin. There is also frequently a febrile reaction, usually of not more than 100° F., rarely amounting to 103° F., developing within twenty-four hours after injection and subsiding within forty-eight to sixty hours. This is accompanied by dry tongue, thirst, and a rapid heart, and occasionally considerable cardiac depression. A frequent observation in secondary syphilis, particularly when roseola is present, is a temporary intensifying of the eruption after the injections, due to a reaction which is attributed to the liberation of endotoxins by the remedy. This reaction usually occurs promptly after injection and then gradually subsides. Urticarial or scarlatiniform eruptions occasionally occur up to ten days after the injection.

Serious complications from the injections have been rare. Many observers report no serious by-effects in large numbers of cases. It is frequent for the total quantity of urine to be much reduced—to 400 or 500 c.c. Suppression of urine in 3 cases of 14 treated, in 1 case lasting nine days, has been reported by Bohac and Sobotka. Transient albuminuria occurs in rare cases. Disturbances of the reflexes have in a few cases been observed. In a few cases temporary scotoma has occurred, usually on the day of injection. Spiethoff has had in two cases sudden total blindness, lasting, however, for only a few minutes. In the first case in a tabetic this occurred fifty hours after injection. In the second case, eight weeks after injection, the patient, healthy except for syphilis, developed sudden blindness in the right eye and ptosis of the lid of ten minutes' duration. Persistent ill effects on the eyes have not been reported.

Spiethoff had one fatal case in a cachectic, anæmic woman of twenty-eight, with tertiary lesions in the throat. She had an injection of 0.5 gm. in the afternoon, followed by 0.01 gm. of morphin in the evening, and was found dead in bed the next morning. The autopsy showed no evidence of arsenic poisoning, and Ehrlich attributes this death to shock in a feeble patient from the local pain of the injection. Fraenkel and Grouven's fatal case from arsenic poisoning has already been referred to.

At present the judgment as to the Ehrlich-Hata remedy must be that it produces remarkable specific effects upon the manifestations of syphilis. Whether it will fulfill all that it promises is not yet established, and can only be determined by time—a very considerable time. It is still in the stage of an experiment, an experiment that has marvelously interesting results but that has not yet established the sure position of the remedy. In the meantime, we are not justified in failing to avail ourselves of the long established benefits which are derived from mercury. Neisser's position, stated in June, is still wise. He thinks the remedy will be specially useful in cases in which mercury has failed, or cannot be used, and he advises against its use in private practice until there is more certainty regarding dosage and the number and repetition of the injections. And before embarking upon the enthusiastic use of this agent the practitioner may well ponder the following advice from the editorial columns of the *Journal of the American Medical Association:*

"Finally, physicians should remember that this new discovery is, after all, an arsenic preparation. In the past all new arsenic preparations, though at first recommended as wonderfully active, and marvelously free from the toxic effects of arsenic, have in the end been found to possess in varying degrees the potency for harm common to this element."

of the initial lesion, or the initial lesions and contiguous glands, or by very vigorous early systemic treatment with mercury. In clinical experiments these methods have been ineffective. The effect of early excision in experimental syphilis has been uncertain, and in the light of the facts in experimental syphilis it would seem to be better to treat the local lesion by applications of mercury, particularly by the inunction of thirty-three-per-cent calomel ointment. This local attempt at abortive treatment, if it does not abort the disease, will not confuse the diagnosis by preventing the characteristic evolution of its symptoms, and is, therefore, not open to the objections next to be considered, which obtain against the early internal use of mercury.

By administering mercury during the primary period the chancre can be made to heal rapidly, but there is the vital objection to the use of mercury at this time that it may prevent the normal evolution of secondaries, and thus render the diagnosis uncertain. This, as has been seen, is a possibility which should not, except for very good reasons, be risked. The use of mercury should not be started until the diagnosis of syphilis is fully established.[1] A positive diagnosis of syphilis cannot be made until sufficient time has elapsed for the development of adenopathy and of cutaneous lesions. Even an authority with so great an experience as Fournier has recorded a case in which what he thought was a typical initial sclerosis with typical enlargement of the inguinal glands did not prove to be syphilis. In the light of such a fact, no one can assume with assurance to make a positive diagnosis of syphilis from the chancre and adenopathy alone. Occasionally a case occurs in which other considerations outweigh the disadvantages of confusing the diagnosis; such cases, for example, are chancres that threaten serious local damage, as about the eye; chancres in the late course of pregnancy, where every effort must be made to prevent infection of the fœtus; and possibly cases where the existence of the lesion for its usual period renders almost unavoidable the infection of innocent persons. Upon the appearance of secondaries the diagnosis may be established beyond any doubt. Then it is time to begin the vigorous management of the case.

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[1] At the present time the positive diagnosis of syphilis from the chancre can be strongly approximated, if not made, by the demonstration of the spirochæta pallida in the secretion with the dark-ground illuminator, and many now advocate the beginning of vigorous treatment upon the demonstration of the spirochæta pallida in these lesions. I have for two years been demonstrating the pale spirochætes in initial lesions. They are, in my opinion, morphologically distinct, and their presence is confirmatory evidence of great importance in considering the character of any suspected initial lesion. The fact remains, however, that the interpretation of spirochætes from their appearance alone is open to mistake, and I am not yet convinced that it is proper practice to begin treatment in these cases until at least we have a characteristic adenopathy in addition, or, better still, adenopathy and beginning eruption. It is excessively important to make the diagnosis beyond the possibility of doubt in any beginning case of syphilis, and while the tendency is to take all of he present findings of experimental syphilis as final and to begin treatment at the arliest moment, it is not established that anything is lost by the wait until the inical diagnosis can be made positive before beginning treatment.

General Management.—The constitutional treatment of syphilis, to be properly carried out, requires at least two to two and a half years. During the first several months of this time the patient is a source of possible infection of others; during the last year or more he is likely to be free from all symptoms, and to doubt the necessity for treatment. In order, therefore, that the treatment may be carried out successfully, it is practically necessary for the physician to have the confidence and coöperation of his patient. To this end it is desirable, upon the establishment of the diagnosis, to give the patient a frank statement of the situation, and to outline the course of the disease and its treatment. If this can be done and the patient is intelligent, the subsequent management of the case usually offers little difficulty. If, for any reason, it is impossible to do this, or if the patient is not intelligent enough to grasp the situation, it is almost the invariable experience that the treatment cannot be carried out satisfactorily through the necessary period.

At the beginning the patient should be warned of the danger of spreading the disease. Particular stress should be laid upon the danger of contagion from mucous patches in the mouth, which he is likely to overlook. In married patients, if the family is to be properly protected, it is almost necessary for the infected person—nearly always the husband—to tell his partner. If he will not do this, he should be required to accept the responsibility for the dangers of the situation. An almost equally complicated social situation is offered when an engaged person acquires syphilis. The only safe course to pursue is to break the engagement, or at least postpone it for several years. If a man will not do this, it is better not to accept the responsibility of his case.

General Treatment.—At the outset and during the course of active syphilis, every effort should be made to maintain the general condition of the patient at as high a point as possible. The more nearly he can live an ideal physical life in the open air and the more completely he can avoid the anxieties and stress of active life the better. He should have plenty of fresh air and sunlight, sufficient but not excessive exercise, a simple, nutritious, and liberal diet, and plenty of sleep. Dissipation and excesses are especially costly during syphilis. In severe cases every effort has to be made to build up the patient. In some cases it may be necessary to provide for a maximum amount of easily assimilated food, and here milk is our best resource.

The question of the use of alcoholic liquors during secondary syphilis is often a difficult one to settle. In general, I believe it may be said that the effects of alcohol are not particularly bad; that is, they are about the same as they are in other infectious diseases, or where syphilis is absent. A regular drinker is a poor subject for syphilis, as he is for any other infectious disease, but in the case of such a patient harm is likely to be done if the use of alcohol is abruptly stopped. The best course to pursue is to diminish, as much as practicable, the amount habitually taken, and especially to prevent periods of excessive drinking. In those who do not use alcohol habitually it is best, under most circumstances, to stop its use entirely. At times, however, malt liquors and light wines are useful in whipping up a failing appetite.

As to tobacco, there is a peculiar reason rendering its use injurious, and that is its unfavorable influence upon the condition of the mouth. It is very likely to irritate mucous patches and to exaggerate their extent, and for this reason it is highly desirable that no tobacco, in any form, should be used during the secondary period of syphilis. Occasionally, unless this is done, it is impossible to control the development of mucous patches in the mouth.

It must be said that many syphilitics, if they receive a proper amount of mercury, and some without any treatment, pass successfully through the secondary stage without attention to any of the matters of right living referred to above. In some cases, however, the health fails badly under secondary syphilis, and then the closest attention has to be paid to these details.

During the course of secondary syphilis the urine and the blood should be examined regularly, the one to discover a possible nephritis, the other to determine the amount of anæmia. The anæmia of secondary syphilis should always have attention. For this anæmia and the cachexia, in addition to mercury general tonics are indicated. Of these iron is most frequently indicated, and is given in the majority of cases with advantage. Strychnin and quinin are also of frequent benefit, and Taylor highly recommends the fluid extract of coca. Prescriptions for tonics recommended by Taylor are:

℞ Ext. erythroxyli cocæ fl.,
Tinct. cinchon. comp.,
Tinct. gentian. comp., } āā.................... f℥ij.

Two teaspoonfuls in wineglassful of water t.i.d., an hour after meals.

℞ Strychnina sulphat. .... gr. i;
Acid phosphoric. dilut.......................... f℥iij;
Liq. pepsinæ, q. s............................ad f℥iv.

One teaspoonful in water after meals, and before going to bed.

Where a sedative is needed for the nervous symptoms of syphilis, hyoscyamus is useful. Any of these preparations can, of course, be given in conjunction with mercury.

Use of Mercury.—The curative treatment of syphilis, so far as it is medicinal, depends upon the use of mercury. Mercury may be administered by the mouth, by inunctions, by subcutaneous injections, or by fumigation. Administration by the mouth is convenient, and when intelligently conducted meets every requirement in the great majority of cases, and it is the usual method of treatment followed in the United States and Great Britain. Inunctions and injections are used more largely in continental Europe. Administration by the mouth is not so vigorous a method of treatment as the use of inunctions or so definite as subcutaneous injections, so that it is well to fortify the usual treatment by the mouth by periods of inunctions, and in critical situations it is best to use hypodermic injections.

The form in which mercury is administered does not make so much difference provided it is assimilated by the patient; this fact is shown by the many salts which have been used. The forms ordinarily used are the protiodide, bichloride, calomel, blue mass, gray powder, with some following for other salts, such as the tannate and the salicylate. The protiodide (dose, $\frac{1}{6}$ to 1 grain t.i.d.) is probably the most eligible salt for administration by the mouth; the bichloride (dose, $\frac{1}{20}$ to $\frac{1}{8}$ grain t.i.d.) is largely used and is an efficient salt, but it is liable to upset the digestion, and unless taken in pill form leaves a very disagreeable metallic taste; calomel and blue mass (dose, $\frac{1}{2}$ to 1 grain t.i.d.) are apt to cause catharsis and salivation. Next to the protiodide, the tannate (dose, $\frac{1}{2}$ to 1 grain t.i.d.) is probably the most satisfactory salt. For combination with the iodides, the biniodide (dose, $\frac{1}{30}$ to $\frac{1}{16}$ grain t.i.d.) is used. It, like the bichloride, is an irritating salt.

The administration of mercury by inunctions is a vigorous method, and may be relied upon in emergencies. It is usually administered in the form of the official fifty-per-cent mercurial ointment, and in my experience the ointments with other bases offer no advantages. If it is irritating to the skin, or too stiff, mercurial ointment may be thinned with vaseline before rubbing in. The beginning dose should be from 30 to 45 grains, or, if there is haste, 60 grains, and the dose may be increased to 90 grains. It should be furnished the patient with each dose in a separate large capsule or waxed-paper packet.

Inunctions are ordinarily given once daily, occasionally less frequently. In giving them care has to be used to avoid hairy surfaces, as dermatitis is much more readily produced around the hairs. For that reason the parts usually selected are the inner sides of the thighs (2), either side of the abdomen (4), of the chest (6), and of the back (8), the palms and soles (9), and the flexor surfaces of the forearms (10).

This provides surfaces for an inunction on a different part for ten successive days. The inunctions are applied to these different surfaces in turn in order to avoid undue irritation. Before the inunction the part should be washed with warm water and soap, and it is well to sponge with one-per-cent carbolic-acid solution. The ointment should be rubbed in slowly without undue friction, and the rubbing should be continued until the ointment has entirely disappeared; at least twenty minutes should be given to the process. Except for the inunctions on the back, they can be carried out by the patient himself, but it is very much more satisfactory to have it done by a professional rubber or nurse.

The administration of mercury by intra-muscular injections has a large number of advocates. It is the method of administration of preference in syphilitic emergencies. Both soluble and insoluble salts of mercury are used for subcutaneous injections. The use of insoluble salts for hypodermic injections is not unattended by danger. They may cause severe mercurial poisoning, from which death has more than once occurred, as it has also from the injection entering a vein and causing an embolus. The preferable method of hypodermic injection is by the use of soluble salts. Many salts have been tried. The most satisfactory are the bichloride and succinamide. The dose of either of these for subcutaneous injection is from $\frac{1}{8}$ to $\frac{1}{4}$ grain daily, or every other day. They are given in one-per-cent solution in water, the bichloride with an equal quantity of sodium or ammonium chloride to insure solution. The bichloride is more painful than the succinamide; on the other hand, it is not easy to get a good solution of the succinamide. The usual practice is to make the injection within the muscle, plunging the hypodermic into the skin vertically. This must be done with scrupulous aseptic precautions, and if any failure in these occurs there is the likelihood of a deep-seated abscess. For this reason I prefer to give the injection hypodermically. In doing this care must be used to give the injection into the subcutaneous tissue and not into the body of the corium. If the injection is made into the body of the corium—a fact which can readily be ascertained by the resistance which the corium offers to the injection—there is likely to occur a small dry slough, which disappears very slowly, but gives no considerable trouble. The best sites for hypodermic injections of mercury are the buttocks, the shoulders, and the back. They may also be given in the abdominal wall, but cause more induration and more tenderness.

Use of Arsenic.—The experimental study of syphilis has led to a renewal of the use of arsenic in its treatment. The views of Neisser upon this subject may be regarded as a fair statement of the subject. He has used it extensively in monkeys, and more recently in men. In many cases it cures symptoms; in others it fails. It is inferior to mercury and is not to be substituted for it. His views are based upon its subcutaneous administration, and he prefers as a preparation, arsecetin; next after that, atoxyl. He regards sodium cacodylate as useless. Welander, from a large experience, has the same view as to the relative value of mercury and arsenic. There is no reason to believe that arsenic should ever be substituted for mercury in the treatment of syphilis.

Use of Opium.—In giving mercury by the mouth, especially with the use of calomel and blue mass, it was formerly a common practice to give with it opium in order to control diarrhea. Ordinarily with care a sufficient amount of mercury can be taken by the patient without the use of opium, and the objections to its use are so many and so important that it should be avoided absolutely, if possible. If it must be given, the best method is to have the patient take just enough paregoric to control the diarrhea; but even this is to be used only with the greatest circumspection. The administration of bismuth may be of distinct service in controlling diarrhea from mercury, and this, with attention to the diet, is usually all that is necessary. If, with the careful administration of mercury by mouth, digestive disturbances persist—a very unusual occurrence—it is better to turn at once to inunctions or injections.

The Iodides.—Mercury is the essential specific remedy in the treatment of secondary syphilis. There is some difference of opinion as to the necessity for the administration of the iodides during the first year of the disease. It is probable, however, that the iodides hasten the elimination of syphilitic toxins, and also of mercury, so that they render the mercury's action more intense; and many excellent authorities, like Taylor, recommend their use at intervals along with mercury during the active period of secondary syphilis.

The general indication for the use of the iodides is the occurrence of gummatous or ulcerative lesions, and there is no difference of opinion as to the value of iodides wherever tertiary lesions of syphilis occur. This applies whether they are precocious and occur during active secondary syphilis, or late in the disease. The iodine salt generally used is the iodide of potassium; occasionally the iodide of sodium, or still less frequently the iodide of strontium, is used as a substitute. Numerous preparations, most of them organic preparations of iodin, have been offered as substitutes for potassium iodide. As Taylor put it, "none of them has shown any reliable therapeutic effects."

The dose of potassium iodide varies from 5 to 10 grains t.i.d. to as much as 2 ounces or more daily in critical situations. There is a strong tendency in America to use the iodides in enormous doses, and in critical situations this is undoubtedly justified by the results. There is no difficulty in the use of very large doses of potassium iodide for periods of a few weeks. In the treatment of most cutaneous lesions, however, it has certainly been my experience that moderate doses—10 to 30 grains t.i.d.—usually leave nothing to be desired, and that it is necessary to exceed such doses only in rare cases. This does not apply to visceral lesions, especially lesions of the nervous system. The iodides are very likely to upset the stomach, and frequently produce the well-known symptoms which are described as iodidism. They usually, also, produce an iodide acne, and occasionally, after long administration, other cutaneous lesions. The iodide acne can well be neglected; it can be controlled in part by the astringent applications used in ordinary acne. To avoid iodism and disturbance of digestion the iodides should be given largely diluted in water and about an hour after meals, when digestion is at the height of its activity. The most satisfactory way to administer iodides is to furnish the patient a watery solution containing one grain of iodide to the drop, and have him take the prescribed number of drops in at least a pint of water. If very large doses are required, it is well to put the entire dose for twenty-four hours in half a gallon or more of water and instruct the patient to drink the total quantity in the twenty-four hours. Another good vehicle in which to take the iodides is milk. The iodides may be prescribed in bitter tonics, such as tincture of gentian, compound tincture of cinchona; other common vehicles are sirup of orange peel, sirup of sarsaparilla, sirup of licorice. Nothing disguises the disagreeable taste of the iodides better than sirup of licorice. Even when given with these, it should be largely diluted with water.

Plan of Treatment of Syphilis.—It is important to treat syphilis vigorously in the secondary stage. If the treatment can be carried out thoroughly for the first six months, it is very unlikely that the patient will have further trouble. For this reason treatment should be started as soon as secondaries appear, and continued without intermission for six months. It is sufficient, as a rule, to begin with mercury by the mouth unless there are urgent indications. It is well to begin with a moderate dose, say ⅙ grain protiodide t.i.d., and to increase this ⅙ grain a day up to a dose of ½ or ¾ grain t.i.d., or until there is slight evidence of salivation, or until it is evident that the evolution of the disease is being rapidly controlled. It is not safe to stop short of ½ grain of protiodide t.i.d. unless there are evidences of hydrargyrism or distinct indications that the disease is being brought under control. If the dose is carried to the point of producing slight ptyalism the quantity should then be reduced ⅙ to ¼ grain, to a point where it is found that the mercurial symptoms disappear, and the dose should then be held at this quantity.

After the treatment is established by the mouth in the way indicated, it should be practically continued, with perhaps short intervals of rest for two years. Under such treatment the patient, in the vast majority of cases, regains his normal condition, and shows no ill effects either from the disease or from the mercury. If he has any evidence of its effect, it is an unusual sense of well-being or a greater regularity in the movement of the bowels.

Some authorities recommend the continuous use of mercury during the first two years; others recommend intervals of rest of about a month each, so that in the first year mercury is taken in the aggregate for about nine to ten months and during the second year in the aggregate for about eight months. But as it is rarely the case that a patient takes mercury absolutely without intermission, the differences in these two methods are not really material. Formerly an occasional syphilographer advised the administration only when symptoms of syphilis are present. This is undoubtedly a dangerous course to pursue, and should never be advised.

It is important to know that the patient is getting a sufficient quantity of mercury to control the disease. In the start, therefore, it is desirable to carry the dose until the first physiological effects of mercury are produced. If the patient's bowels and teeth are in good condition this may be relied upon as a somewhat larger dose than is necessary for his efficient treatment, and the dose may be reduced from a quarter to a half of the full physiological dose, and maintained at this point. More important even than the determination of the patient's tolerance of mercury as evidence that he is having a sufficient dose is the course of the disease. Rapid disappearance of the evidences of specific toxæmia, a feeling of well-being, an appearance of good health, the rapid disappearance of the eruption, the gradual subsidence of the sclerosis at the site of the initial lesion and of the glandular enlargement, the improvement in the anæmia, normal sleep and appetite, and the maintenance or regaining of the normal weight, are the best evidence that he is getting a proper amount of mercury.

The treatment during the entire two years may be carried out by the administration of mercury by the mouth. If emergencies arise, inunctions or injections should be used; and it is a good rule, even when the greater part of the treatment is given by the mouth, to remit the use of mercury by the mouth for periods of two to four weeks, and during this time to give inunctions. One may use inunctions at the outset of treatment, or after the first month of treatment by the mouth give a series of inunctions for twenty or thirty days, and then return to the administration by the mouth. At the end of six months, if the patient is doing well, it is a good plan to stop treatment for two weeks to a month, while the patient is under observation. If this does nothing else, it reassures the patient to find that he gets along well without medicine. Then begin again for a second period of from three to five months, and so end the first year.

Ordinarily there are no definite indications in the evolution of syphilis for the use of potassium iodide during the first year, but no harm is done and possibly some good results if moderate doses of potassium iodide are combined with mercury for two or three periods of a month each during the first year.

During the second year the internal treatment should be kept up in the same way as during the first, with intervals of two weeks to a month without treatment every three or four months. It is desirable during the second year, in order to forestall any possible gummata, to give mixed treatment at least part of the time, say one month in every three of treatment, using some such combination as the following:

℞ Hydrarg. biniodidi	gr. i–ij;
Potassii iodidi	ʒss–i;
Aquæ	℥i;
Sir. aurantii cort. vel.,	
Tr. cinchonæ comp.	℥iij.

One dram, t.i.d., an hour after eating.

If treatment is carried out for more than two years, the same general plan should be followed during the third year, meeting emergencies, if they arise, by the vigorous use of mercury and the iodides. Emergencies that arise later than the second year demand the iodides, and if they are rebellious the iodides should be given in large doses. They may be sufficient without combination with mercury, but the treatment is more efficient if the use of the iodides is combined with that of mercury. This applies to all gummatous lesions no matter how late in the course of syphilis they occur.

There is some difference of opinion as to how long the systematic treatment of syphilis should be continued. Many French authorities, following Fournier, advise three to four years. Taylor holds that at the end of the second year a patient may be pronounced cured, provided he has been vigorously treated and the treatment has consisted of inunctions and the use of iodides in conjunction with mercury by the mouth, and provided he has had no cutaneous lesions within a year or more, the lymphatic engorgement has disappeared, and he is in good physical condition. Most American syphilographers accept two to two and a half years as sufficient. There is, however, no objection to the administration of mercury and the occasional administration of the iodides for three years or more; and in order to err on the safe side, I prefer that treatment should be continued for at least three years, and that during each of the two succeeding years—making five in all—the patient should have at least two periods of mixed treatment of four to eight weeks at intervals of four to six months.

Our reliance against gummatous lesions rests in the iodides, with mercury as an adjuvant; practically all of the so-called alteratives, like sarsaparilla, are of no specific value, and can be safely neglected. There is only one preparation for which an exception should be made, and that is that old example of polypharmacy called Zittman's decoction. There is enough undoubted testimony to the value of Zittman's decoction in very severe rebellious tertiary lesions to warrant one in remembering it as a resource in rare rebellious cases of tertiary syphilis.

Local Treatment of the Chancre.—Ordinarily the chancre is an entirely trivial ulcer, which requires little local treatment beyond cleanliness and the application of a dusting powder. It should be irrigated twice a day with a weak antiseptic solution, such as bichloride 1:1,000 or 1:5,000, carbolic acid 1:200 or 1:500, boric acid, or hydrogen peroxide one to five or six of water. After this the lesion may be dressed with dry powder, such as calomel, boric acid, aristol, or europhen. If the lesion is where it will be macerated, or if there is considerable secretion, it is better dressed with a small wet dressing, such as weak bichloride or carbolic or boric-acid solution. For this purpose one of the best applications is black wash or yellow wash, which is best dabbed on the surface for a few minutes, allowing the powder to settle, and then followed by a small dressing of cotton wet with the solution. Caustics should not ordinarily be used upon chancres. If there is a mixed infection, or if the lesion is phagedenic, it should be treated in the way suggested for soft chancre, and if caustics are used they should be strong antiseptics, like carbolic acid, or caustics vigorous enough to destroy all of the diseased tissue. In infected lesions iodoform is the best local application.

Treatment of the Syphilides.—The nonulcerative syphilides ordinarily require no local treatment. They speedily subside under constitutional treatment. If for any particular reason it is especially desirable to hasten the disappearance of secondary lesions it can be done by local application of preparations of mercury, usually in the form of ointments. Secondary syphilides upon the face should be given local treatment in order to hasten their disappearance. For this purpose a mercurial preparation should be applied freely at night and less freely in the morning. The best preparation is ammoniated mercury in proportion of 15 grains to a dram to the ounce of ointment. Another good application is solution of bichloride 1:500 or 1:250, in alcohol or cologne. Both of these applications are also beneficial for the seborrhœa which frequently complicates the facial lesions. Other local applications are five-per-cent oleate of mercury, or mercurial ointment, one or two parts to the ounce of other ointment, but these applications are not as good as those first given.

Ulcerative Lesions.—The ulcerative gummatous lesions, whether they occur in the secondary or in the tertiary stage, should be locally treated, first by careful irrigation with an antiseptic solution, of which bichloride 1 : 5,000 to 1 : 1,000 is, I think, the best. This irrigation should be repeated at least twice a day and oftener, if necessary to keep them clean. After irrigation the lesions should, if possible, be dressed with wet dressings, of which bichloride 1 : 5,000 or 1 : 10,000 is one of the very best. After the ulcers heal the wet dressings and irrigation may, of course, be stopped and the further involution hastened by the local application of mercurial ointment or ammoniated mercury ointment. If the application of wet dressings is not practicable, the ulcers should be irrigated and dressed with antiseptic powders or with dressings spread with ammoniated mercury ointment, or mercurial ointment half strength. These plans of treatment are frequently advocated as the plans of preference, but they do not provide for the removal of the secretions as wet dressings do, and they are not so satisfactory.

In extensive ulcerating syphilides it is an excellent plan to give the patient daily general baths in bichloride solution 1 : 30,000 or 1 : 15,000; that is, one or two drams of bichloride with an equal amount of ammonium chloride or hydrochloric acid to thirty gallons of water.

Mucous Patches.—In order to prevent as far as possible the development of mucous patches and to give mercury satisfactorily, the mouth should have careful attention. The teeth should be cleaned twice daily with the aid of a dentifrice,[1] and the mouth rinsed with an antiseptic solution, such as Dobell's solution, or the following:

℞	Potassium chlorate	ʒi;
	Sirup or honey	℥ss;
	Water ad	O.i.

To this may be added tr. of myrrh, ℥ss.

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[1] A chlorate of potash dentifrice is one of the best. A good formula for such a dentifrice I have found to be the following:

Pot. chlorate	40 parts
Soap	10 "
Prep. chalk	15 "
Powd. orris	2 "
Water	17 "
Glycerine	14 "
Sugar	2 "
Acid carbolic	.15 parts
Ol. gaulth. et ol. cassia	.8 "

℞	Boric acid	℥ss;
	Glycerine	℥ss;
	Oil of cassia	♏v–x;
	Water ad	O.i.

Better than any of these as a mouth wash, if mucous patches are abundant, is solution of bichloride 1:5,000; but this is disagreeable, and blackens the teeth.

If mucous patches are present, they should be touched every day or every few days with a saturated solution of nitrate of silver or one- to two-per-cent chromic acid, and when they are abundant the use of the mouth wash should be frequent.

Mucous patches are not likely to develop about the folds of the body and around the genitals and anus if the parts are kept clean. They may be avoided by the use of dusting powders to prevent maceration. If they occur the parts should be kept scrupulously clean, washed twice daily with weak bichloride solution, the patches dusted with calomel or some other antiseptic, and occasionally painted with two- to four-per-cent solution of silver nitrate.

Condylomata.—The treatment of condylomata is the same as that for mucous patches. Neither mucous patches nor condylomata should be dressed with ointments.

Alopecia.—Syphilitic alopecia usually yields only to internal treatment, but some good, perhaps, can be done by the use of a vigorous stimulating application, such as bichloride 1:500 or 1:1,000 in alcohol.

PALMAR AND PLANTAR SYPHILIDES.—These are the most rebellious to treatment of syphilitic lesions, whether they occur in the secondary or tertiary stage. They may be quite uninfluenced by internal treatment. Locally they require stimulating applications, as vigorous as can be borne without causing an acute inflammatory reäction. For this purpose ammoniated mercury or mercurial ointments in varying strengths are usually most efficient, and with them it is often desirable, for its stimulating effect, to combine tar. Before the application of ointments the parts should be thoroughly washed with hot water and soap to remove the scales. Over the fissures which occur, and frequently as the sole application over the entire surfaces of the palms and soles, an excellent application is bichloride in solution in tincture of benzoin in the strength of 1:1,000 to 1:250. This is to be painted on the surface twice a day, and allowed to dry on.

Congenital Syphilis.—Active syphilis in the mother should be vigorously treated during pregnancy, both for the effect upon the mother and for the effect upon the child. After the birth of the child every possible effort should be made to maintain its nutrition at the highest point. It should, if possible, be nursed, but it can be nursed only by its own mother; this must never be overlooked. If the child is born with active evidence of syphilis, vigorous treatment should begin at once. If it is born apparently healthy, specific treatment should not be started until the disease manifests itself, for it is possible that the child has escaped infection.

The constitutional treatment of hereditary syphilis is upon the same principles as that of the acquired form. It should be vigorously pushed, and if the child survives should be continued for the same period as in acquired syphilis. Children as a rule bear relatively large doses of mercury. The best treatment is usually mercury by the mouth or by inunctions. Protiodide, in doses of $\frac{1}{20}$ grain t.i.d., increased rapidly if well borne, may be used, but is apt to cause diarrhea. The better forms for children are calomel or gray powder, in doses of from $\frac{1}{10}$ to $\frac{1}{2}$ grain t.i.d.; gray powder can frequently be given in doses of 1 grain t.i.d., without causing diarrhea. Calomel and gray powder are best given by mixing with sugar of milk and placing dry upon the tongue just before nursing. If diarrhea occurs it may be controlled with paregoric, and the use of this in infants is much more justifiable than in adults. For inunctions, mercurial ointment in the dose of 15 to 20 grains, increased to 30 grains if the child does well, may be used daily. It is customary to apply the mercurial ointment to the swathing cloth and have it rubbed in by the motions of the child, but it is, I think, better to have it rubbed in over different areas of the body in the usual way. Rarely hypodermic injections of bichloride or succinamide, in doses of $\frac{1}{32}$ to $\frac{1}{8}$ grain daily, are used. Potassium iodide is not usually distinctly beneficial in congenital syphilis, which is active secondary syphilis, and is likely to upset the digestion, but it may be given in doses of $\frac{1}{2}$ to 2 grains t.i.d.

The local treatment of the lesions is the same as in acquired syphilis. The use of weak bichloride baths, $7\frac{1}{2}$ to 30 grains to 8 gallons of water, is of good service in extensive suppurating congenital syphilides.

Special care has to be taken to keep the folds of the body clean. This is done by attention to cleanliness, and, if the parts are not diseased, by bathing them with water, followed by alcohol, and then by a dry dusting powder. The mucous patches and condylomata in the folds of the body are to be treated with great care in the manner indicated for the treatment of these lesions in acquired syphilis. Particular attention should be paid to the nose. It should be cleaned frequently, and every possible effort made to overcome ulceration within the nostrils.

XVII.—TUBERCULOSIS OF THE SKIN[1]

Tuberculosis plays a double rôle in the causation of skin diseases: (1) It may occur locally in the skin; (2) in addition there are a number of affections in which, with more or less plausibility, tuberculosis is invoked as the indirect cause through the action of tuberculous toxins circulating in the blood.

Tuberculosis itself appears in the skin in the form of lupus—the typical form—miliary tuberculous ulcers or nodules, erythema induratum, and scrofuloderma. The lesions which it produces in the skin are, with minor variations, of the same character as those produced elsewhere. They are granulomata with the usual structure of infective granulomata. Tubercle bacilli are found in them, but as a rule sparsely because of the unfavorable conditions for their growth which the skin presents, and inoculation tuberculosis can be produced from the tissues.

The so-called tuberculides (Darier) or paratuberculoses (Johnston) are not tuberculous deposits in the skin, but are inflammatory lesions which are attributed to the influence of tuberculous toxins present in the blood. The grounds for this assumption are by no means thoroughly established, although in many of the conditions the association with tuberculosis in other structures is so constant as to be very suggestive. The paratuberculoses are:

(1) Lichen scrofulosorum.
(2) Acne scrofulosorum.
(3) Acne agminata.
(4) Acnitis or folliclis.
(5) A scarlatiniform erythema produced by tuberculin.
(6) Eczema scrofulosorum.
(7) Pityriasis rubra of Hebra.
(8) A dischromia like the pigmentary syphilide.
(9) Lupus erythematosus.

In the case of lichen scrofulosorum, acne scrofulosorum, and scarlatiniform erythema from tuberculin injections the evidence of the causative importance of tuberculous toxins is very strong. In the rest of the affections in the list the evidence is purely presumptive and the relationship doubtful.

Lupus vulgaris is the typical, and by far the most frequent, form of

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[1] Discussion, *Trans. 3d International Congress of Dermatology*, 1896, Crocker, Hallopeau, Hyde, Hutchinson, *et al.*—Boeck, C., *Archiv*, vol. xlii, 1898.—Johnston, "The Cutaneous Paratuberculoses," *Phila. Month. Med. Jour.*, February 1899.—*Trans. Inter. Congress Derm.*, 1900, "Les Tuberculides," Boeck, C. Fox, Darier, *et al.* —Fox, C., *Brit. Jour. Derm.*, vol. xiii, 1900.—Hartzell, *Amer. Medicine*, vol. viii, 1904.

sis in the skin, and deserves the first consideration. The para-
ses, which are toxic disturbances, are considered in the several
which they most naturally fall, and are not taken up here
e infectious diseases.

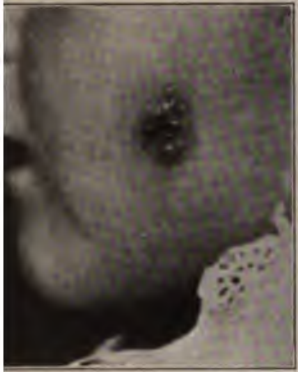
XVIII.—LUPUS VULGARIS[1]

YM: *Lupus.*

(Lat., *lupus*, a wolf) is a chronic neoplastic infiltration of
roduced by the tubercle bacillus and characterized by brownish-
cles deeply embedded in the skin which coalesce with the for-
f nodules and patches, and which upon disappearing, either
ithout ulceration, leave scars.

erm lupus was originally applied to any spreading ulcer of the
r it was applied to ulcer of the leg. Willan and Bateman, in
ing of the nineteenth century, first applied it to approximately
al entity which it now designates.

the term lupus alone is used it always signifies lupus vulgaris.
ythematosus, the other disease of whose name the word lupus



UPUS VULGARIS. Single small
eek in young child. (Author's
.)

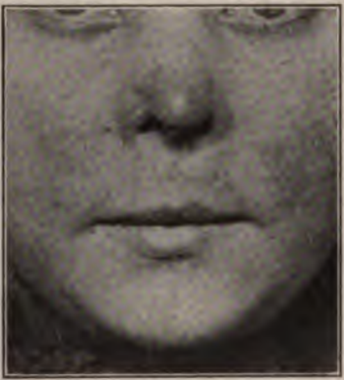


FIG. 172.—LUPUS VULGARIS.
(Author's collection.)

is a distinct disease of doubtful if any relationship to lupus

tomatology.—The primary lesion of lupus is a miliary papule
bedded in the skin. This is essentially a minute infective

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e, J. C., *Boston Med. and Sur. Jour.*, Nov. 12, 1891.—Lespinnes, *Jour. mal.*
ober 1891.—Dubois-Havenith, "Du Lupus vulgaire," Brussels, 1890
sive monograph).—Morris, *Lancet*, October, 1904, "Treatment of Lupus

granuloma and its course typifies the entire disease. This prima is a pinhead to small pea-sized brownish-red or yellowish papul embedded in the corium and showing on the surface as a smo tening spot. to use Hut now classical sion, like a "apple jelly" ded in the ski a true neopl filtration, an the redness pressed out, t tumor still sh der pressure a ly embedded speck. An patch of lu consist, not of one, but of several or many of these small a tubercles. At the beginning they may be slightly depressed with the skin, but they grow slowly, and in the course of months they develop into small nodules which are of semitranslucent brownish-red color, sometimes soft and sometimes of moderately firm consistence, and readily break down under force. The further course of these nodules is variable. They may persist for weeks or months without change; in some cases they undergo fatty degeneration and are absorbed without ulceration, leaving shiny, wrinkled, white scars. In other cases they break down with the formation of indolent, roundish, irregular ulcers with fl borders and an unhealthy, red, granulating base, from which purulent secretion which dries upon the surface into dirty

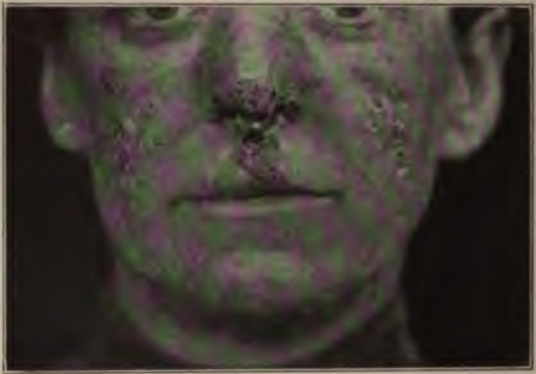

FIG. 173.—LUPUS VULGARIS. (Author's collection.)

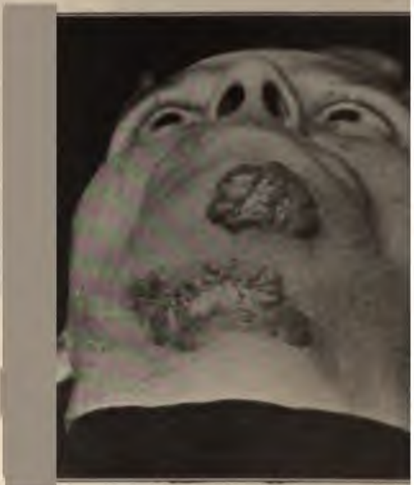

FIG. 174.—LUPUS HYPERTROPHI (Author's collection.)

crusts. Such an ulcer may consist of a single broken-down , but as a rule it is formed from the coalescence of numerous ing nodules, and in the contour of the ulcer the fact of its pro- i by the coalescence of adjacent nodules will be apparent. Fur- idence of its composite character will be found in the existence rete satellite tubercles around the border. The individual ulcer- esions and the larger ulcers tend to heal spontaneously with the ion of firm, fibrous

Where the lesions erficial the scars may tively thin and parch- e, but in the larger eper ulcers they are nd firm. After scar is formed there is a y for tubercles to in it, and these may their ordinary course of involution without ion or of ulceration. e have a combination nentary factors: of lly nodules which do erate, of others which own, and of scar tis- ich is thick and dis- , and in which the tends to recur.

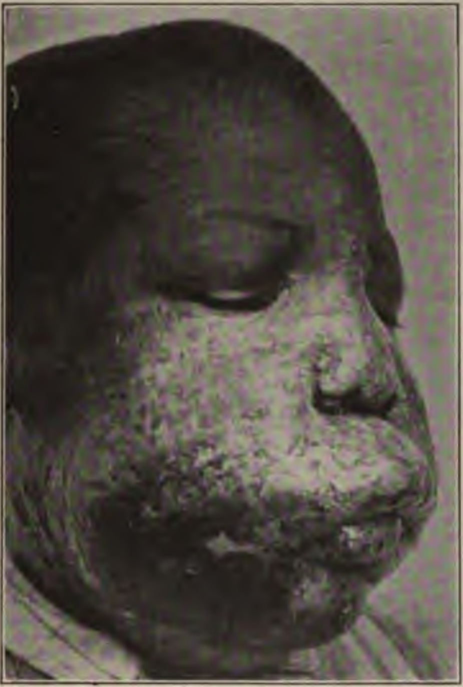

FIG. 175.—LUPUS SCLEROSUS. (Engman and Mook's collection.)

combinations in g and in evolution lupus patches may many different clini- ures. Many of these are so characteristic that they are given names, but they are all variations of one disease and are essen- he same. Most of these special names are inserted below, but for convenience of description and for the identification of the

disease may occur in patches of closely aggregated but distinct s. In such a case we will have a dull red, inflammatory patch, more or less with discrete tubercles which may persist as either elevated scaling tubercles or may form minute ulcers. At times ons occur as discrete tubercles or groups of tubercles, which may ly distributed over the body (*l. disseminatus seu discretus*).

More frequently the lesions coalesce into nodular plaques, which are dull or brownish red, with a sharply elevated, firm, nodular border and a softer nodular center. Such a patch may persist for a long time with more or less scaling, and perhaps with a tendency to involution at the center. Or it may develop into heaped-up masses of nodules which form a distinct tumor (*l. tumidus or l. hypertrophicus*). Again, these nodular patches may break down with the formation of ulcers of characteristic type. In another type of hypertrophic lupus there is great thickening of the tissues from lymphatic obstruction and resultant œdema. This occurs especially on the upper lip. In these cases, as a result of lymphatic œdema or of a long-continued inflammatory process, there may be excessive formation of connective tissue, producing dense and exaggerated permanent thickening of the parts (*l. sclerosus*). This fibroid thickening of lupus may occur in small patches where, as a result of the chronic inflammatory process, the lesions have an unusually firm stroma of connective tissue. In such cases the nodules may have the appearance of those of ordinary lupus, but they are dense and hard and resistant to the curette, unlike the easily broken-down tissue of the ordinary tubercle. Such lesions are most common to the buttocks, and occur less frequently on the face. They are usually resistant to treatment.

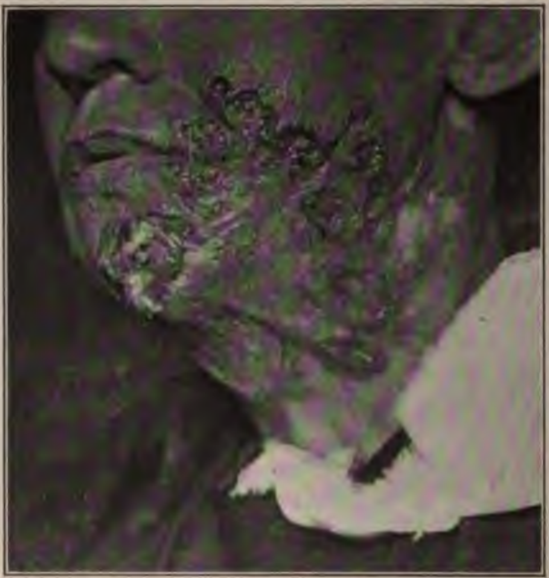

Fig. 176.—Lupus Serpiginosus. (Author's collection.)

Not infrequently in lupous ulcers, probably as result of secondary infection, there is the formation of exuberant granulation tissue and an overgrowth of papillary vegetations, producing papillomatous and warty dry or suppurating lesions (*l. papillomatosus or l. verrucosus*[1]).

Sometimes the ulcerative lesions spread serpiginously with the formation of polycyclic ulcerating borders, while there is a tendency to involution and the formation of scar tissue at the center, forming lesions which closely resemble ulcerating syphilides (*l. serpiginosus*).

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[1] Morrow, *Amer. Jour. Cut. Dis.*, vol. vi, 1888.

In very rare cases lupous lesions take on an arrangement in patches which undergo involution at the center and leave a border of active disease at the periphery. The border may be nodular without ulceration, but is more frequently ulcerated and crusted, with scar tissue behind it (*l. annularis*[1]).

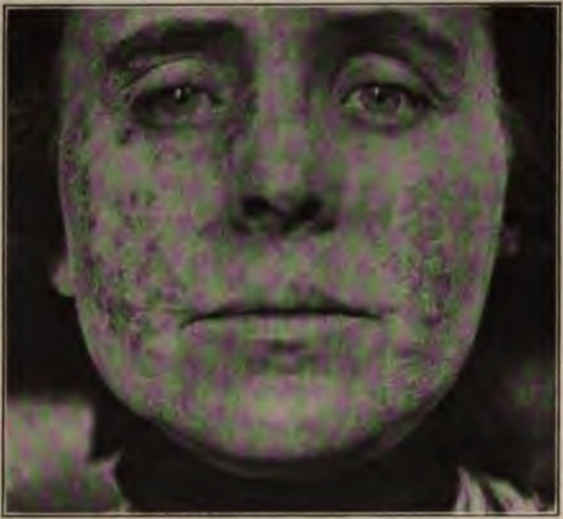

FIG. 177.—LUPUS VULGARIS ERYTHEMATOIDES. (Author's collection.)

In patches where the disease remains as flat or slightly elevated tubercles without ulceration there may be considerable scaling both of the tubercles themselves and of the scars from recent tubercles which have subsided (*l. exfoliativus*). Where, as is occasionally the case, the lesions are without elevation, and scale freely, there may be a superficial resemblance to psoriasis (*the lupus psoriasis* of Hutchinson). The resemblance, however, is only very superficial. The scaling occurs on top of typical lupous infiltrations in the skin, and there is usually more or less typical scarring.

Sometimes the patches of lupus spread centrifugally without ulceration with the formation of patches which are dry, bright or dark red in color, more or less scaly, with a sharply defined, slightly elevated border, and with a center perhaps slightly depressed (*l. vulgaris erythematoides*[2]). Such cases may resemble very closely lupus erythematosus, but always there are present in the border, or as satellites, the typical miliary, apple-jelly tubercles, which appear yellowish under pressure. Indeed, these characteristic tubercles, occurring either in the border or as satellites around it, are to be found in all of the different varieties, and furnish the most important clew to their character.

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[1] Elliot, *Amer. Jour. Cut. Dis.*, 1896.

[2] (5) Leloir, *Jour. des mal. cutan.*, vol. iii, 1891.—Hardaway, *Trans. Amer. Derm. Assn.*, 1893.

The most frequent site for lupus is the face, especially around the tip of the nose, and the face is usually involved if the disease exists upon other parts.[1] In addition it not infrequently appears upon the forearms and legs, the buttocks and the outer sides of the thighs, and no part of the body is exempt, although it rarely, except by extension from adjacent areas, involves the scalp, the middle of the forehead, the upper lids, the neck, the palms, the soles, or the genitals.

The extent of the disease is exceedingly variable. It may exist only as a single patch not larger than a small pea. It most frequently, perhaps, appears as a single area of disease, but with almost equal frequency it occurs in two or more patches, and occasionally it is very widely distributed. The disease is not markedly symmetrical, but where it is extensive there is usually a rough symmetry, and sometimes this is quite definite.

Lupus is ordinarily very indolent. It progresses slowly, and the entire course of a single patch may extend over many years. It may have periods of quiescence, followed by periods of renewed activity, and so it may go on for many years in a progressively destructive course. On account of the lack of pain and its insidious growth it may produce an amount of disfigurement before its grave character is discovered which in the end is quite surprising to the patient. Quiescent periods may rarely last for years, and the disease may become active without discoverable reason. It is apt to be excited by external irritation or depressing internal influences. In very rare cases the disease spreads rapidly, with active hyperæmia and other evidences of acute inflammation and with rapid destruction of tissue (*acute lupus*).

In late years the frequency of its occurrence upon the mucous membranes of the mouth, and especially of the nose, has been emphasized. Audry, who has looked for lesions of mucous membranes very carefully, has found them on the nasal mucous membrane in every instance of lupus of the face. In Copenhagen, where Finsen has had a large opportunity to study lupus, involvement of the mucous membranes of the nose or mouth occurs in seventy to eighty per cent of the cases. Bender[2] found in 380 cases involvement of the mucous membranes in 45.5 per cent. In 6 cases it existed on the mucous membranes alone; in 46 cases it had apparently begun on the mucous membranes; the nose was involved in 115, the conjunctivæ in 21, the lachrymal duct in 24, the lips in 43, the palate in 31, the tongue in 1, the larynx in 13, the rectum and vulva in 1.

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[1] In 374 cases Bender (*Deutsch. med. Wochenschr.*, 1886) found the location of lupus as follows: face, 287; generalized over the face, 115; nose, 70; cheek, 35; nose and other parts of the face, 25; nose and lips, 15; lips, 16; temple and forehead, 6; chin, 3; eyelids, 2; arms, 40; legs, 15; face and neck, 12; face and extremities, 6; face and arm, 3; ear, 3; general, 2; scalp, 2; hand and foot, 1; nape of neck, 1; back, 1; palate, 1.

[2] "Ueber Lupus der Schleimhäute," *Archiv*, vol. xx, 1886. (Bibliography.)

In rare instances the primary pinhead-sized or larger reddish tubercles covered with silver-gray exfoliating epithelium may be found upon the mucous membranes. These may coalesce into larger dull grayish plaques resembling syphilitic mucous patches. More frequently the lesions in the mouth and nose show as indolent, easily bleeding, granulating ulcers. In the nose they are apt to be just within the nasal orifices; in the mouth, on the inner side of the lips and on the gums. In the mouth, papillary overgrowths, forming fungating ulcers, are common. The ulcerating lesions also appear upon the hard and soft palate and pharynx, and may involve the larynx. The tongue is very rarely involved. In the nose the lesions may develop primarily or extend from the adjacent skin, the disease persisting in the form of chronic ulcers, and at times causing perforation of the cartilaginous septum. Lupus may occur on the conjunctiva primarily, but more frequently by extension from the cheek, or perhaps through the lachrymal duct. The cornea sometimes becomes involved with production of perforation, and panophthalmitis and loss of the eye have been seen. Lupus may involve the external auditory canal and cause perforation of the drum membrane. In very rare cases the middle ear may be involved, usually by extension through the Eustachian tube.

In the slow spread of the disease it gradually involves the deeper tissues and destroys the soft parts. It also attacks adjacent cartilage, but usually does not cause destruction of bone unless as a result of secondary infection or of the development of true tuberculous osteitis.

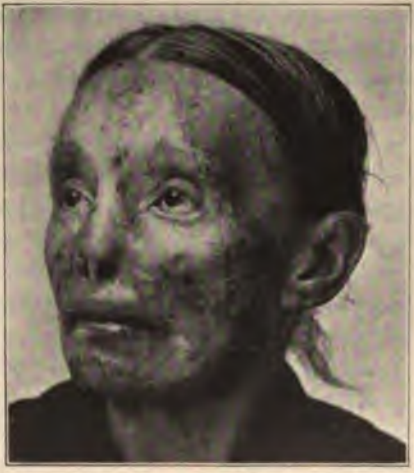

FIG. 178.—LUPUS, SHOWING TYPICAL SCARRING OF FACE IN CASE OF LONG STANDING. (Schamberg's collection.)

The amount of disfigurement which lupus causes depends upon the extent and duration of the disease. The disfigurement is greatest about the face. Here, if it is long existent, the nose usually shows the greatest effects; but there may be ectropion and distortion of the mouth, and there is more or less scarring wherever the disease is located. After long-continued extensive lupus of the face, the bandlike scars which are formed may convert the face into a hideous mask of itself.

The subjective symptoms in lupus are practically nil. The ulcerative lesions are slightly tender, but spontaneous pain is absent. There are no constitutional symptoms. In extensive cases there may be anæmia and more or less general cachexia. This is usually not a result of the lupus, but more often of the constitutional dyscrasia which accounts for the presence of the lupus or of tuberculosis of deeper tissues. Lespinnes has described four ulcerative cases in which, before an exacerbation of the disease, there was a febrile reaction, followed by prostration, with gastrointestinal and bronchial catarrh, endarteritis and other serous inflammations, and, in some of the cases, the production of organic heart lesions and the later development of general tuberculosis. These cases, however, are unique.

Complications.—In the majority of cases lupus remains confined to the skin and subcutaneous connective tissue and the adjacent mucous membranes. It may be complicated by tuberculous abscesses and gummata, periosteitis and osteitis, with caries and necrosis of the bone. Involvement of the glands adjacent to lupus foci is rare, but occasionally occurs, and in some cases there occurs persistent tuberculous lymphangitis. Infection with the common pyogenic cocci is, of course, the rule in the ulcers, and attacks of erysipelas sometimes occur, but not commonly.

As a result of the long-continued chronic inflammatory process, perhaps with repeated attacks of erysipelas, and the resultant obstruction of the lymphatics and blood vessels, elephantiasis of the parts may occur.

An occasional complication in lupus scars, not in the lupus lesions themselves, is epithelioma. According to Leloir,[1] this occurs in about two per cent of cases.

Fordyce[2] has reported the development of an endothelioma.

Etiology and Pathology.—Lupus is rare in the United States. It is common in Great Britain and on the Continent of Europe. No class is exempt, but it is seen most frequently among the poor, and apparently its distribution is directly associated with the depth of poverty of the lowest classes. About two thirds of the cases occur in women.

It has a marked tendency to begin in childhood, usually between three years of age and puberty, but it may be seen in the first year of life, and, in rare cases, has begun in old age—as late as sixty-three years in two cases of Crocker's and at forty-six in another. Of 96 hospital cases of Colcott Fox's, 5 began in the first year, 30 before the fifth, over half before the tenth, 6 between the thirtieth and fortieth, and 4 after the fortieth.

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[1] *Brit. Jour. Derm.*, 1890 (abstract).—Desbonnets, Paris, 1894, "Epitheliome et Lupus." (Bibliography.)

[2] *Amer. Jour. Med. Sci.*, August, 1900.

Lupus may occur in persons who are in good health, but as a rule it is seen in those who are below standard in general vigor. When existent, it is apt to be worse in winter, or when the patients are physically depressed from any cause. In a few cases it has been observed to become active during pregnancies and lactations, and to subside between these periods, but these conditions exert no regular influence upon its course. There is fairly definite relationship between lupus and the tuberculous diathesis. A tuberculous family history is found in a fair proportion of cases (thirty-three and one third per cent, Bender), and the existence of tuberculosis in other tissues is a common association. In 38 cases of Besnier's, 8 had pulmonary tuberculosis, and in the combined statistics of Bender, Colcott Fox, Block, and Sachs, in a total of 514 cases of lupus, 346 presented evidences of past or present tuberculosis of other tissues. Occasionally disseminated lupus follows acute specific fevers, especially measles, the explanation probably being either that the patients are vulnerable to tuberculous infection during convalescence r that during their illnesses tuberculous glands or other foci of the sease break down with the liberation of tubercle bacilli in the cir-lation.

There are many cases of lupus on record in which the evidence of rect external inoculation is very strong. The number of ways in which has been transmitted are almost innumerable: by kissing, by tattooing, the ritual circumcision of the Jews, by piercing the ears; in laundry omen, by washing the linen of tuberculous individuals; in butchers, y handling tuberculous meats; in persons who have to do with tuber-ulous cadavers; by contact with abrasions, by accidental inoculation of uperficial wounds, as in herpes zoster, and, very rarely, vaccinations. According to Leloir, the methods of inoculation of the skin in lupus are as follows:

(1) Directly from without.
(2) By extension from deeper tuberculous foci.
(3) By extension through the lymphatics or veins.
(4) Through the blood.
(5) *In utero.*

The first and second are most frequently its origin.

When lupus originates from external inoculation it most frequently develops in the form of tuberculosis verrucosa cutis, but typical lupus may develop from such inoculation. The type of lupus is influenced by several facts:

(1) Method of infection:
  if from within it is likely to be nodular;
  if from a local infection, verrucose or ulcerative.
(2) Possibly the abundance of the tubercle bacilli and the extent of contamination with common pus organisms.
(3) Individual resistance:
  in the cachectic and strumous it is apt to be ulcerative and destructive;
  in those of stronger resistance it occurs most frequently in the form of nonsuppurative lesions.

Histology and Bacteriology.—As was first shown by Koch, the disease is due to the effects of the tubercle bacillus and its toxins. Bacilli are demonstrable in the tissues only with difficulty, many sections often having to be examined before a bacillus is found, even in the active border of the lesions. The bacilli are indistinguishable from those of other forms of tuberculosis; inoculation of lupus tissue in animals produces typical tuberculosis, and the use of tuberculin causes a violent local and general reaction.

The lesions of lupus are histologically variations of the typical nodules of tuberculosis. They may develop in any part of the corium from the papillary layer to the subcutaneous tissue, usually along the course of a vascular or lymphatic channel. The individual nodules making up a lesion may have a limiting border of connective tissue, and so continue separated by intervals of entirely or comparatively healthy tissue; or they may become confluent, or be so surrounded by abundant infiltration of embryonal cells as to be indistinguishable, forming a veritable neoplasm of embryonal and plasma cells; or, again, the individual nodule may spread by irregular peripheral extension along the routes of the vascular and lymphatic channels.

The schematic type of tubercle—giant cells, zone of epithelioid cells, zone of embryonal cells—is only exceptionally met with in lupus; one or another of its constituents may either be lacking, or in itself constitute almost the whole of the lesion. The giant cells of lupus are usually very well developed, of large size, and with many—ten to a hundred—nuclei. The appearances of phagocytosis in the giant cells are probably attributable to accidents of formation. The giant cells usually occupy the central portion of each nodule, and are surrounded by embryonal lymphoid or epithelioid cells, and the character of the nodule varies according to which of these predominates. The lymphoid tubercle occurs always in the immediate neighborhood of a blood or lymph vessel, and is composed of a few or no central giant cells surrounded by cells entirely similar to lymphocytes, though considered by many to result from proliferation of the fixed connective-tissue cells. They are closely pressed together in consequence of rapid multiplication, and may be surrounded by a connective-tissue capsule, or spread along the lymphatic channels. The epithelioid nodules often contain a very large number of giant cells of large size, surrounded by numerous plasma cells, and usually around this epithelioid mass there forms an accumulation of lymphoid cells. An indefinite reticulum may often be made out running through the cellular masses of both forms. Central caseation and diffuse necrosis are almost never seen in lupus, though colloid and myxomatous degenerations have been described. As the surrounding cellular infiltration becomes dense, interfering with the nutrition of the central cells, fatty degeneration and disintegration occur, with subsequent absorption or ulceration.

While the tissue between the tubercles may in the nodular form of the disease be almost free from changes, in other cases there is a sharp reaction to the virulence of the pathogenic agent, and the nodular accumulations are no longer recognizable on account of the diffuse infiltration, composed of large plasma cells disseminated through the orium and mingled with lymphoid cells, connective-tissue nuclei, the nore resistant elastic tissue, and newly formed vessels. Giant cells ccur sparsely, and are usually to be found toward the periphery in the ess dense areas of the neoplastic infiltration. Evidences of œdema and erous imbibition are marked. The whole of the tissue may show a oincident general hyperplasia, causing an elephantiasis, or this may e restricted to the papillary layer of the corium and a verrucose strucure result.

The fibrous tissue in the immediate vicinity of the foci is destroyed, he elastic fibers being more resistant than the white, and being visible requently as isolated fibers or collections of strands within and just round the infiltrations. More peripherally there is a tendency to foration of thick cicatricial tissue. The walls of the vessels in the vicinity re affected; the endothelial cells become loosened and proliferate, tendg to obliteration of the lumina; the outer coats are increased in thickess, and frequently infiltrated with lymphoid cells.

The effect on the epithelium takes the forms of both atrophy and ypertrophy. In the nondiffuse, nodular forms with slight lymphoid filtration and dense connective-tissue hyperplasia, the epithelium is reatly thinned, being represented by perhaps only three or four rows f cells; papillæ and interpapillary processes are lacking, and the cells ay even lose their staining reactions and become charged with pigment.

In the cases of diffuse infiltration, however, and doubtless from the ame causes that produce the rich increase in connective-tissue elements, he epithelium proliferates; the stratum mucosum is thickened, the interpapillary processes elongate, branch, and penetrate throughout the infiltration of the corium, with formation of cornified globules within the epithelial processes. This hyperplasia of the epithelium may become so marked as to be almost indistinguishable histologically from epithelioma.

Under the influence of the œdema which is present, especially in the instances of rich, diffuse, highly vascular infiltrations, and as a result sometimes of secondary infection, the cells of the epidermis often become swollen, serum collects between them, small abscesses form between the corium and epidermis, the corneous layer or even the whole of the epidermis becomes dislodged, and there results coagulation of the serous exudation upon the surface, with formation of crusts, and at times ulceration.

The sebaceous, hair, and sweat follicles usually atrophy and disappear under the pressure of the infiltration surrounding them. Occasionally they become separated from the epidermis and persist as isolated epithelial masses; or they may proliferate and become fused with the other epithelial hyperplasia.

Diagnosis.—The pathognomonic feature of lupus is the peculiar apple-jelly nodules which are embedded in the skin. These can be distinguished from a simple inflammatory papule by the fact that the color of the inflammatory papule is entirely obliterated by pressure, while the infiltration of lupus under pressure leaves a yellow spot which is still visible. This is best seen under pressure with glass. Other characteristic features of the disease are its beginning in childhood, its slow and painless but progressively destructive course, and its predilection for the face, especially the tip of the nose.

Lupus is most apt to be confused with ulcerating syphilide, epithelioma, and lupus erythematosus. The diagnosis from these several diseases is considered with them.

Lupus is distinguished from scrofuloderma in that the process of scrofuloderma is essentially subcutaneous, and consists of broken-down, suppurating tuberculous glands connected with the skin by ulcerative sinuses. The skin itself is red and inflamed, and shows an unhealthy inflammatory reaction, but there are no typical apple-jelly tubercles of lupus.

Ringed forms of lupus might occasionally resemble leprosy, but in leprosy there is entire absence of the apple-jelly nodules of lupus; there is anæsthesia in the confusing patches, usually enlargement of the ulnar nerve, and the patches increase much more rapidly than in lupus. Further, there are apt to be the characteristic changes of leprosy about the face and a history of residence in a leprous locality.

Occasionally patches of nonulcerative scaling lupus may resemble uperficially patches of psoriasis. The scaling is much less, is not fine nd silvery, but occurs in large, flaky scales, underneath which there s the soft brownish, typical infiltration of lupus. The lesions are very ndolent, and whether ulceration occurs or not leave some scarring, vhich is entirely absent in psoriasis.

Scaling lupus might also resemble scaly patches of eczema. In a scaling patch of chronic eczema the scaling is more abundant, the base of the lesion is a chronic inflammatory process with a good deal of leathery induration, there is entire absence of the typical nodules and of scarring, and there is usually more or less itching.

In doubtful cases of lupus, injections of Koch's old tuberculin may be employed as a diagnostic measure; at first 2 milligrams may be injected, and the dose may be increased subsequently to 5 milligrams, or ven 10 milligrams. A 10-milligram dose may produce a local reaction n an erythematous lupus, but it produces none in syphilis or epitheli-ma. In leprosy and scrofuloderma, tuberculin produces a reaction, o that it is of no diagnostic value in these conditions, and in leprosy r extensive tuberculosis the reaction may be very violent.

Prognosis.—The prognosis in lupus, as to both permanency of re-ults and extent of disfigurement, is much better since the methods of reatment with radiant energy have come into vogue. The prognosis epends upon the extent of the disease, the health of the individual, e depth of involvement of the tissues, and the extent of destruction hich has already occurred. The disease is very apt to recur after pparent cure, and cases should be carefully watched, and when recur-nce occurs promptly treated.

Treatment.—The general management of lupus patients is along e established lines of treatment of other forms of tuberculosis. Every-ing possible should be done to build up the general health of the atients; attention should be paid to hygienic methods of living, with articular stress upon an abundance of fresh air, outdoor life, if possible, nd a liberal, nutritious diet. The digestion should be carefully guarded nd maintained at as high a degree of efficiency as possible. As in ther forms of tuberculosis, cod-liver oil is the most useful tonic, but ther tonics may be required to meet general indications. All measures nding to increase the resistance of the individual are useful as adju-ants in the treatment of lupus, but it must be said that lupus very rely yields to such measures alone.

The internal specifics are few; Crocker and Pringle highly recom-mend thyroid extract, as suggested by Bramwell, but to be useful its dministration has to be continued for a year or more. At the beginning grains of the desiccated thyroid is given daily; after two weeks he dose may be increased to 10 grains, and in two weeks more, if there are no symptoms of thyroidism, to 15 grains; but the increase should be made cautiously, and only after an interval of at least two weeks.

Recently McCall Anderson has called attention to excellent results in the treatment of extensive cases of lupus from injections of tuberculin T.R., and previously there had been various reports of its value as an adjuvant in the treatment of lupus. In rebellious cases it is worthy of trial. The new tuberculin—tuberculin T.R.—is used, beginning with a dose of 1 to 2 milligrams, and this is increased by the addition of 2 milligrams to each subsequent dose. A maximum of 20 milligrams has been given in some cases, but such doses are not advisable, for, besides being dangerous, where good effects are obtained from tuberculin they usually result from the early smaller doses. In giving tuberculin the attempt is made to produce a very slight reaction without causing an elevation in temperature of more than 1° F. When the reaction is slight and the doses small the injections may be repeated every two days. If the dose is large the intervals should be much longer, and if the reaction is acute the doses should not be repeated until it has subsided.

Local Treatment.—The local treatment of lupus has been revolutionized by the introduction of radiotherapy, including under this term both Roentgentherapy and phototherapy. The exhibition of patients cured of lupus by Finsen at the International Congress of Dermatology in Paris, in 1900, marked an epoch in the treatment of the disease. The technique of the treatment of lupus by phototherapy and by Roentgentherapy has been given briefly under general therapeutics. Both methods give successful results. Finsen's method is tedious where the disease is extensive, and is not applicable to lupus of the mucous membranes or to ulcerative patches, because in such lesions sufficient pressure cannot be exerted. Treatment with Roentgen rays gives the same excellent results, is applicable to any surface upon which direct exposures can be made, and can be applied at one time to large surfaces. For these reasons it is, I think, on the whole, the preferable method of treatment, and in America, at least, is superseding Finsen's method.

While carrying out radiotherapy or any other method of treatment, lupus patches which are ulcerating or which present open surfaces should be kept clean and treated according to ordinary surgical principles, with the view to overcoming or preventing secondary infection. Where there is much ulceration or free suppuration the surface should be irrigated once or twice daily with an antiseptic solution, and should be dressed in the mean time with compresses of boric-acid solution or of weak bichloride or some similar antiseptic. Where there is much suppuration it may be more convenient to use, instead of wet dressings,

dressings of antiseptic ointments spread on gauze. For such purposes one can use ointments of carbolic acid, one of the less irritating mercurials such as ammoniated mercury, iodoform, aristol, resorcin, or other similar antiseptics. In patches which are acutely inflamed it may be desirable for a while to use bland soothing dressings before beginning radiotherapy. Excellent applications under such circumstances are wet dressings of calamine lotion, lead-and-opium wash, and aluminum acetate solution.

Before the introduction of radiotherapy the radical treatment of lupus consisted in either chemical or mechanical methods for destroying the diseased tissues. These methods still have a certain field of usefulness, but fortunately it is much restricted.

Irritating local applications may be used for the purpose of actually lestroying the lupus nodules, or for the purpose of exciting an acute nflammatory reaction and in this way indirectly destroying the diseased issue. A favorite application of this sort is mercurial ointment, or, etter, mercurial plaster. White recommends strong applications of ichloride, 1 or 2 grains to the ounce, which may be applied continuously ither in solution or in ointment. Salicylic acid, a dram to the ounce, ay be used in the same way; better than this is Unna's strong salicylic-cid plaster. Another method of applying salicylic acid is to dissolve in collodion in the strength of ½ to 1 dram to the ounce; resorcin is sed in the same way. Pyrogallic acid is one of the favorite irritating pplications. It may be used in a ten-per-cent ointment, applied two three times a day for a week, and then followed by a mercurial plasr. It is also used in a ten-per-cent solution in collodion; with this it is sual to combine ten-per-cent salicylic acid. When any of these applicaons are used they should be applied continuously until an acute inammatory reaction is produced, and then bland dressings used until e reaction subsides.

Still stronger applications may be used for the immediate destrucon of the lesions. Thus the nodules may be destroyed by the applicaon of caustic potash, of chloride of zinc, or of arsenical paste, or by ring them out with the nitrate-of-silver stick. The use of the nitrate-silver stick is largely mechanical, and a method quite similar is boring to them with a dental burr or similar instrument.

The surgical methods of treatment of the lesions are excision, curetteent, scarification, and cauterization.

All except excision depend upon the principle of removing part of e diseased tissues and at the same time exciting an acute inflammatory action. Excision of lupus lesions is a practical method where the sease is young, and the operation can be carried well beyond the border disease without too much destruction of tissue. The great objection to excision is the great danger of recurrence, and it is not at present, in my opinion, an eligible method of treatment.

In curetting the lesions a small spoon is used which can scoop out the smaller nodules. After curettement of lesions it is usual to touch the surface with pure carbolic acid or with a dilute solution of caustic potash or chloride of zinc, or some similar caustic application.

Linear scarification of the patches should be done first in one direction in lines close together, and then these should be crossed by other lines at right angles, the aim being to destroy as many blood vessels as possible and at the same time to produce an acute inflammation. Linear scarification is not so radical as curettement, and is apt to give smoother, more pliable scars. As a substitute for scarification, multiple puncture with a sharp-pointed knife is sometimes used, but has no advantages over scarification.

The same object that is obtained by curettement or scarification is attained by cauterization of individual nodules or patches with a small-pointed galvanic or Paquelin cautery. The objection to this method is that it destroys the healthy and diseased tissue with equal readiness.

After any of these applications bland antiseptic dressings are required, and no matter what the method of treatment, repeated and persistent efforts are necessary.

Another method similar in its results to the foregoing, which has some advocates, is the use of electrolysis. In this way the diseased tissue can be destroyed, and it is satisfactory for small lesions.

Recent methods which should be mentioned are the use of high-frequency currents, radium, hot air, and freezing with ethyl chloride.

The use of high-frequency currents and radium has been suggested in the place of x-rays. They can doubtless both be used successfully over small areas, but there is no reason to believe that they have any advantages over better tried methods.

Freezing the lesions with ethyl chloride has been suggested, but has not come into general use. From a limited experience in the treatment of lesions by freezing with solidified carbonic acid, I believe that it or liquid air would likely furnish an efficient and rapid method of treating discrete nodules or small patches of lupus.

Clinical Forms. Lupus Verrucosus (*Tuberculosis verrucosa cutis, Verruca necrogenica*).—Lupus verrucosus occurs in lesions which have an infiltrated but not nodular base, upon which there is a papillary overgrowth.

The lesion of lupus verrucosus is an elevated plaque with an indolent inflammatory base and a warty, papillary surface. The base is fairly firm, diffuse, and not nodular, and there is an absence of apple-jelly tubercles. The extent of the papillary hypertrophy varies very considerably, both in height and in extent. It sometimes entirely covers the entire lesion, but frequently leaves a wide margin of the base uninvolved. There is more or less exudation of pus from the sulci between the papillæ, which dries upon the surface into dirty crusts and which, on pressure, exudes from the surface, and there may be points of well-marked ulceration in the papillary surface. The lesions are very indolent, but tend to spread slowly, with perhaps healing at the center, with formation of scars, and at times with spontaneous healing of the entire lesion.

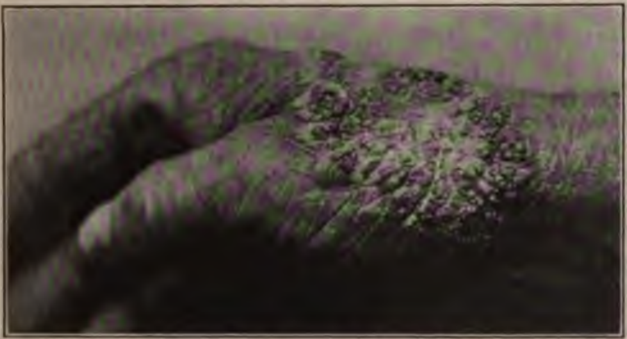

FIG. 179.—TUBERCULOSIS VERRUCOSA CUTIS. (Anthony's collection.)

It occurs usually on the backs of the hands, and next most frequently on the feet. There is usually a single lesion, but the lesions may be multiple.

It is rarer than the nodular form of lupus vulgaris. It is usually produced by local inoculation, and for that reason is seen in persons who have to do with tuberculous materials. As it occurs in butchers and other handlers of meat, it was described by Riehl and Paltauf as tuberculosis verrucosa cutis; the form occurring on the hands of anatomists and pathologists has long been known as verruca necrogenica.

Anatomically it differs from nodular lupus chiefly in the papillary hypertrophy and the greater hyperplasia of the epidermis.

Tubercle bacilli are more abundant in the tissues than is the case in lupus. It is usually described as a separate disease, but it is a lupus, with the same pathogenesis, essentially the same anatomical structure, the same course, and subject to practically the same forms of treatment, and it is more conducive to clearness to regard it as a form of lupus than to erect it into a separate disease.

Lupus Marginatus (*Hilliard's lupus*).—This term has been given by Hutchinson,[1] without definite evidence that the disease is a form of lupus, to a few cases he has seen of an eruption which began with an initial patch resembling lupus verrucosus, apparently due to local inoculation on the hand, and which spread up the arm to the shoulder. Patches similar to the initial patch occur in the course of the disease, and also small cornlike nodules. In other cases the patches and nodules have appeared on the face. The lesions persist indefinitely and yield only to destruction with the actual cautery or caustic potash.

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[1] Hutchinson's "Smaller Atlas," Plates XIII and XIV.

XIX.—MILIARY TUBERCULOSIS OF THE SKIN AND TUBERCULOUS GUMMATA[1]

SYNONYMS: *Tuberculosis cutis vera, Tuberculosis cutis orificialis, Tuberculosis ulcerosa.*

Miliary tuberculosis in very rare instances occurs in the skin. The lesions begin as miliary tubercles which, however, are rarely seen before they have undergone caseation and broken down. The ulcers thus formed are superficial, irregular, round or oval, with soft edges and a base made up of unhealthy yellowish-red granulations. There is a scanty, seropurulent secretion which dries upon the surface in a thin crust. Miliary tubercles may be found in the base of the ulcers and frequently around the borders. The lesions may be single or multiple. They spread slowly, and if multiple they may coalesce by slow extension into larger irregular and perhaps serpiginous ulcers. These tubercles occur about the orifices of the body as the result of extension or inoculation from internal tuberculosis, but in a few rare cases tuberculous ulcers unassociated with internal tuberculosis have been observed.[2]

In very rare cases miliary tubercles and larger tuberculous nodules without ulceration are found in the skin.[3] They may be single or in groups, and while the few cases that have been recorded have been located chiefly about the face, lesions have been observed upon other parts of the body, and unassociated with demonstrable internal tuberculosis. In the same way tuberculous ulcers unassociated with visceral tuberculosis have been observed.[4]

Tuberculous ulcers, as a rule, are very indolent, and pursue a slow but continuous course. In a very few cases they progress rapidly.

The lesions may be painless or very painful.

Tuberculous Gummata.[5]—Occasionally true tuberculous gummata occur in the subcutaneous tissue of cachectic individuals. They begin

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[1] Kaposi, *Archiv*, xliii and xliv, 1898.

[2] DuCastel, *Annales de Derm. et de Syph.*, vol. ix, 1898.—James, *Brit. Jour. Derm.*, vol. v.

[3] Jessner, "International Atlas," Part 13, Plate XXXIX.—Liddell, *Brit. Jour. Derm.*, vol. xii, 1900.

[4] Crocker's "Atlas," Plate LXXII, Fig. 3.

[5] Mracek, "Atlas," Plate LXXXVI.—Ravogli, *Jour. A. M. A.*, 1898.

subcutaneous nodules of normal color, which increase to the size of a
zelnut or olive, and ultimately, as a rule, involve the skin, and then
eak down, though occasionally they are absorbed without ulceration.
hese gummata are indolent and comparatively painless. The ulcers
oduced by their breaking down are sluggish and painless, with indo-
nt, red, flabby borders and unhealthy bases, with a scanty discharge
seropurulent secretion. Exuberant granulations may form and con-
rt the lesions into papillomatous ulcers. These ulcers occur in stru-
ous individuals, and a common location is the legs. These lesions
ay be associated with tuberculous lymphangitis, which in turn may
sult in secondary connective-tissue hyperplasia and elephantiasis.

Etiology and Pathology.—True tuberculous ulcers of the skin are
exceedingly rare affection, although Kaposi, who has had the largest
pportunity of studying the lesions, has seen twenty-two cases. Except
the rarest instances, the lesions are found late in the course of fatal
ternal tuberculosis, usually of the respiratory tract, and are the result
inoculation.

Anatomically the lesions show the ordinary structure of miliary
bercles. Tubercle bacilli occur more abundantly than in any other
taneous lesion, and are easily found.

Diagnosis.—The presence of indolent, painless, progressive ulcers
th flabby borders at the orifices of the body, in association with ad-
nced constitutional tuberculosis, makes the diagnosis easy. When
curring independently of internal tuberculosis the positive diagnosis
uld depend upon the discovery of the bacillus or inoculation experi-
nts.

Prognosis and Treatment.—As they usually occur late in general
berculosis, little is to be done for the lesions except palliative treat-
nt along ordinary surgical lines. When resulting from inoculation
individuals not fatally ill, their treatment is upon the same prin-
les as that of lupus. Kaposi succeeded in curing some of his cases.

XX.—SCROFULODERMA

Scrofuloderma is the term applied to the sluggish, inflammatory,
ppurating lesions of the skin occurring around the sites of sinuses
ich connect with underlying suppurating tuberculous glands or with
ner subcutaneous tuberculous foci. It is thus not a true tuberculosis
the skin.

The skin lesions are due to a chronic inflammatory process which
a part of the inflammation surrounding the suppurating tuberculous
ci. There is an indolent purplish patch which may be pierced by
ly one or two ulcerating sinuses or may be riddled with them. There

may be suppuration of the skin from secondary infection with pyogenic organisms, and in very rare cases there may be tuberculous ulcers in the skin itself as a result of secondary tuberculous infection. Underlying the skin lesions there are found one or more tuberculous glands. Some of these may be broken down and suppurating, others swollen, firm, and painless.

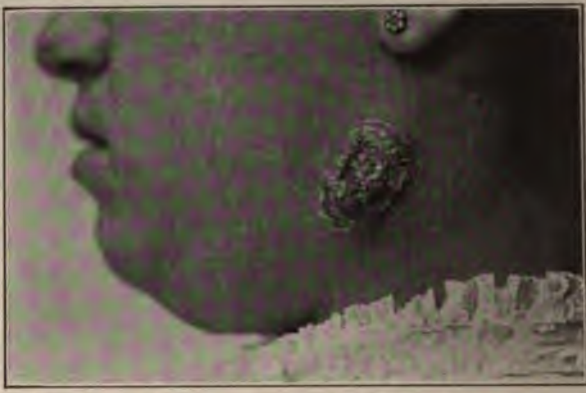
FIG. 180.—SCROFULODERMA. (Schamberg's collection.)

The lesions of scrofuloderma are seen most frequently on the sides of the neck in association with cervical tuberculous adenitis. They are not infrequent in the axillæ, and may occur wherever there is tuberculous adenitis or other subcutaneous focus of tuberculosis which has ruptured through the skin.

The radical treatment is that of the underlying tuberculosis, and is usually surgical. I have cured a good many of the suppurating cases by the use of x-rays.

XXI.—ERYTHEMA INDURATUM[1]

SYNONYMS: *Bazin's Disease, Erythema induratum scrofulosorum, Érythème induré des scrofuleux* (Bazin), *Érythème noueux chronique des membres inférieurs* (Besnier).

Erythema induratum is a disease affecting cachectic and strumous individuals, and characterized by the occurrence, usually on the backs of the legs, of indolent, deep-seated, gummalike nodules which may be absorbed but tend to undergo necrosis with the formation of sluggish ulcers.

Symptomatology.—The lesions begin as deep-seated, diffused, indurated, pea-sized nodules or plaques which are more appreciable to touch than to sight. When first seen they may be bright red, but they later become purplish, with a dark-red or purplish areola. They gradually enlarge in the course of days or weeks to one half to one inch or more in diameter, become doughy and of more ill-defined outline. From this stage they may undergo resolution without ulceration, or necrosis of the skin and subcutaneous tissue may occur with the formation of rather deep, irregular, sluggish ulcers, looking very much like ulcerating syphilitic gummata. The lesions are usually multiple and by their coalescence before ulceration may form brawny, indurated patches, or by coalescence after ulceration form large, irregular ulcers. The ulcers are very sluggish, and may persist indefinitely or spontaneously heal. Both the nonulcerative and the ulcerative lesions, upon disappearing, leave scars — the nonulcerative lesions pigmented atrophic scars, the ulcers pigmented deep scars—which are surrounded by a dark purplish-red, persistent halo.

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[1] Bazin, "Léçons sur la scrofule," 2d edit., 1861.—Besnier, *Annales*, 1889.—Fox, Colcott, *Westminster Hosp. Reps.*, 1888; *Brit. Jour. Derm.*, 1893 (valuable review).—White, J. C., *Jour. Cut. and Gen.-Urin. Dis.*, 1894.—Dade, *Jour. Cut. and Gen.-Urin. Dis.*, 1899 (review and bibliography).—Johnston, *ibid.*—MacLeod and Ormsby, *Brit. Jour. Derm.*, 1901 (review and bibliography).—Whitfield, *Brit. Jour. Derm.*, July, 1905.

The lesions are multiple, and occur typically upon the backs and sides of the calves. Less frequently they occur upon the fronts of the legs, upon the thighs, and upon the arms. Other tuberculous lesions in the skin have been observed in association with them.

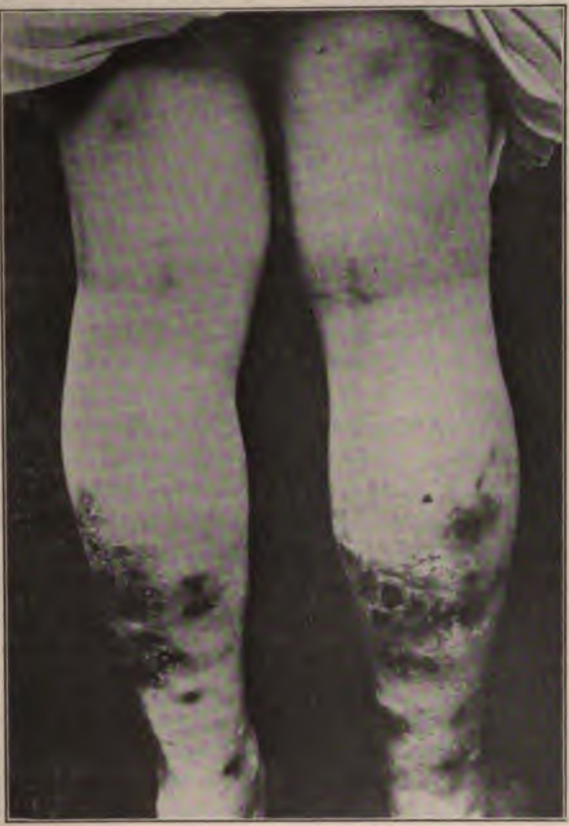

FIG. 181.—ERYTHEMA INDURATUM.
(C. J. White's collection.)

The course of the disease is chronic, and it may recur in successive attacks through many years. Pain and tenderness, as a rule, are absent or slight, but they may be considerable.

Etiology and Pathology.—Erythema induratum is a rare disease. It is seen chiefly among the poorer classes, in those whose occupations keep them on their feet and who have a poor peripheral circulation. It occurs between the ages of twelve and thirty, and most frequently in girls.

Clinically it is closely associated with a tuberculous individual or family history.

The pathological character of the lesions apparently varies in different cases. Thibierge and Ravault, and later Fox and Eyre, found in the lesions the structure of tuberculous infiltrations with giant cells, and by inoculations of tissue produced fatal tuberculosis in guinea pigs. Leredde and Johnston and others, however, have not been able to confirm these findings in their cases. It seems probable, from these diverse findings, that there are two classes of the cases: one, in which the lesions are produced by the tubercle bacilli themselves, another, in which the lesions are the result of tuberculous toxins, or even of depressing factors unassociated with tuberculosis.

Diagnosis.—The characteristic features of the disease are the painless, indolent nodules most apparent to the touch, which tend to break down with the formation of ulcers; the location on the legs, especially on the calves; and the occurrence in cachectic individuals, especially in those with poor peripheral circulation.

The disease is most likely to be confused with syphilitic gummata or with erythema nodosum. The lesions differ from syphilitic gummata in their indolent evolution and course, their usual symmetrical location on both legs, their predilection for the calves, and their failure to respond to potassium iodide. From erythema nodosum they differ in their much slower evolution and much longer duration, in their preference for the backs of the legs, in the absence of distinct pain or tenderness, and in their tendency to break down.

Treatment.—The general management of the cases and the internal treatment consist in the usual measures indicated in strumous cachexia.

The ulcers should be thoroughly cleaned at least once daily with an antiseptic solution, and afterwards dressed with an antiseptic powder or salve. Next in importance to constant cleanliness in the local treatment is the support of the circulation in the legs. Rest with the legs in a horizontal position is of great value. Where the patients must be on their feet, the legs should be supported by gauze bandages over the dressings, or if the ulcers are not numerous dressings of Unna's gelatine jelly may be applied, in which a window is cut over each ulcerating point so that it can be dressed daily.

XXII.—LEPROSY[1]

SYNONYMS: *Lepra, Lepra Arabum, Elephantiasis Græcorum, Spedalskhed.*[2]

Leprosy is a chronic, infectious disease caused by the bacillus lepræ, with symptoms which appear chiefly in the skin or in the nervous system, according to whether the organisms find their chief development n one or the other of these tissues.

Because of its biblical associations, leprosy is a disease of peculiar historical and tragic interest. It can be traced back to the earliest historical times. There is evidence of its existence during the earliest Chinese, Indian, and Egyptian civilizations. Its symptoms were fully and accurately described by Aretæus in the first century of the Christian era. The bacillus lepræ was discovered by Hansen in 1871, and reported by him to the Medical Society of Christiania in 1874.

Leprosy was universal in Europe in the Middle Ages, then subsided in the twelfth, thirteenth, and fourteenth centuries, and has become in the last century a rarity in most of the highly civilized countries of the world. It still exists in Spain and Portugal, Greece, Turkey, the Mediterranean islands, Iceland, Lapland, and the Russian shores of the Baltic. It is found practically throughout Asia and Africa and their adjacent islands. It has been spread among the islands of the Pacific by the Chinese. It is common throughout the West Indies and is found in Mexico, Central and South America. In Canada small foci exist at Tracadie and in two adjoining counties in New Brunswick. There are a few cases in Manitoba among the Icelandic and Norwegian settlers, and it is found on the Pacific coast, both of Canada and of the United States, where it has been imported by the Chinese. The chief focus n the United States is in Louisiana. There are also a few cases among he Scandinavian settlers in Minnesota and the Northwest and numerous cases in Key West. Sporadic cases are occasionally found all over he United States. Dyer estimates that there are over five hundred cases in the United States, the most of them in Louisiana and southern Florida.

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[1] Thin, G., "Leprosy," 1891; Article "Leprosy," *Encyclopedia Medica.*—Leloir, Traité pratique et théorique de la Lèpre."—Hillis, "Leprosy in British Guiana."—usen and Looft, "Leprosy in its Clinical and Pathological Aspects."—Morrow, ticle "Leprosy," *Twentieth Century Pract.*; Article "Leprosy," Morrow's "System of rm.," vol. iii, p. 56.—Dyer, *New Orleans Med. and Sur. Jour.*, October, 1897; "Enmic Leprosy in Louisiana," *Phila. Med. Jour.*, September 17, 1898.—Montgomery, W., "Leprosy in San Francisco," *Jour. A. M. A.*, July 28, 1894.—Jones, *New leans Med. and Sur. Jour.*, vol. v, 1877–78.—Hyde, "The Distribution of Lepy in North America," *Trans. Cong. Amer. Phys. and Surg.*, 1894.—White, J. C., Leprosy in the United States and Canada."

[2] In view of the ignominy—wholly unjustified—which attaches to the name leper, has been suggested by leprologists to refer to the disease by the Norwegian name, Spedalskhed."

Symptomatology.—Leprosy pursues an exceedingly slow course. It has a period of incubation and prodromal symptoms before the positive manifestations of the disease, but both of these are so uncertain and indefinite that we have no accurate knowledge of them. The incubation period is altogether uncertain. The disease may develop within a few weeks after exposure; on the other hand, it may develop many years—five, ten, twenty, or more—after residence in a leprous district.

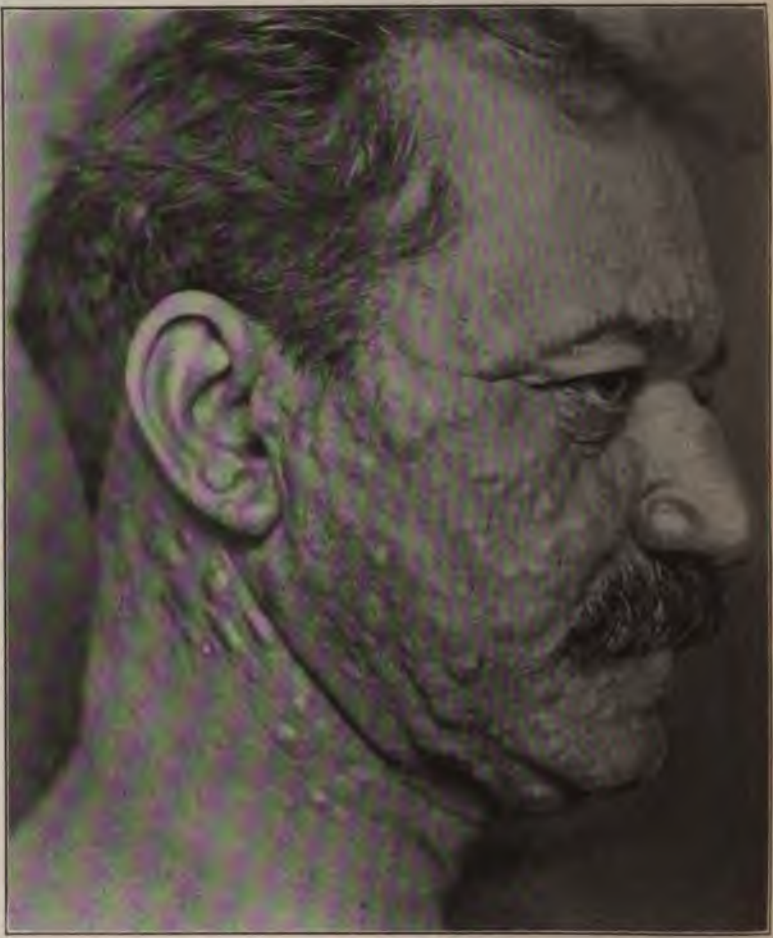

FIG. 182.—TUBERCULAR LEPROSY OF FACE. Early Stage. (Author's collection.)

Leprosy in its manifestations resembles in many particulars syphilis, and there is every reason to believe that it results from inoculation.

It is inevitable, therefore, that the probability of an initial lesion should suggest itself, and that persistent efforts should have been made to discover it. An initial lesion has been sought in the early lesions in the mucous membrane of the nose, in the early macular lesions on the body, and in local manifestations at a known point of injury which occurred while the patient was exposed to leprosy. These efforts to establish an initial lesion have entirely failed, and it is probable that none exists.

Lepra bacilli develop by preference in the skin and nerves. While they may develop in other tissues, the skin and peripheral nerves are the sites of their predominant effects, and the manifestations of the disease are accordingly most pronounced in or confined to these. In some cases the manifestations are almost exclusively the result of invasion of the nervous tissue; in others they are the result of invasion of the skin. Accordingly, leprosy is divided into tubercular leprosy when it chiefly affects the skin, and anæsthetic or maculo-anæsthetic or nerve leprosy when it chiefly affects the nerves. A third variety,

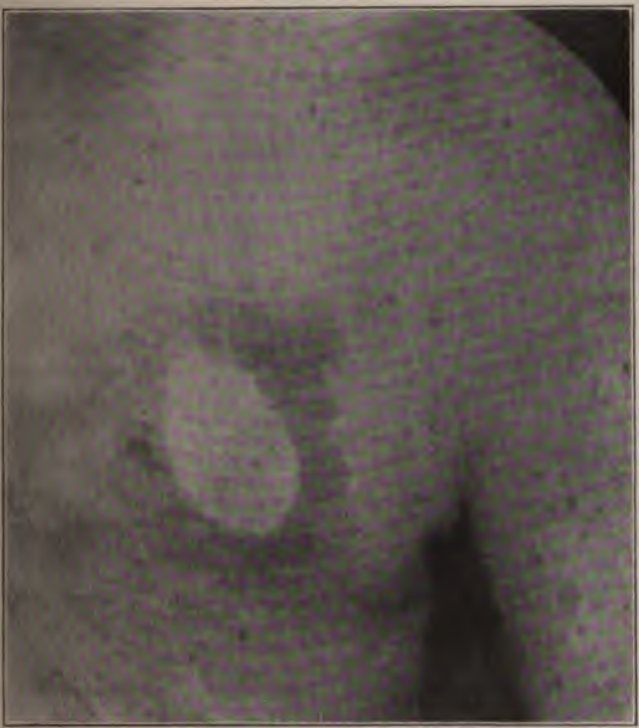

FIG. 183.—BEGINNING PATCH MIXED LEPROSY. Same Case as Fig. 182. (Author's collection.)

mixed leprosy, is also usually described, but all cases of tubercular leprosy sooner or later have symptoms arising from nerve involvement, and become mixed cases.

The symptoms of anæsthetic and tubercular leprosy, in their well-defined forms, are quite distinct. Tubercular leprosy is a more active and severe manifestation of the disease than anæsthetic. Where leprosy is endemic in a severe form, as in places where its occurrence is comparatively recent, the majority of the cases are tubercular; where it is in milder form, as in older foci, the anæsthetic type is equally as frequent or more frequent than the tubercular.

Tubercular Leprosy.—Tubercular leprosy is usually preceded by toxic symptoms such as are found in subacute infectious diseases. These consist of more or less general depression, pains or stiffness of the limbs, rigors, febrile disturbances, with profuse sweating and occasionally epistaxis. The symptoms are quite indefinite, and a diagnosis cannot be made from them alone. As the toxic disturbances increase there occur erythematous patches, with ill- or well-defined borders, of reddish to dark-red color, chiefly upon the face and hands and extremities. These patches vary in size from that of a coin to that of the palm, disappear on pressure, and are apparently toxic erythemas analogous to those found in other toxic disturbances. These patches may entirely disappear or may be followed by slight pigmentation. With the appearance of this

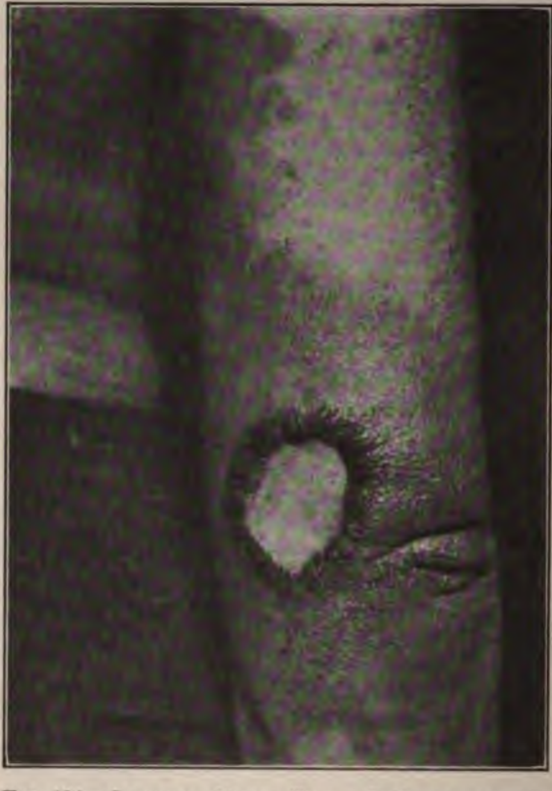

FIG. 184.—LEPROSY, MIXED TYPE. Both the atrophic center and the nodular border anæsthetic. (Author's collection.)

uption the patient's health usually improves, but sooner or later re repeated similar constitutional disturbances, with a recur- f erythematous patches, which finally become permanent and d or mahogany color. There is some infiltration, and they no disappear on pres- After repeated at- f this sort definite infiltrations make ppearance in the , and perhaps also s where there has previous erythema. filtrations appear as odules, which slow- rge to the size of a ea or a hazelnut, or ecome much larger. re oval or rounded, be slightly flattened, to firm consistence, ry in color from a to violaceous or rown. They may crete or confluent, hen confluent they rm heaped-up, con- ate swellings. The tions are situated in the corium and the epidermis remains but from the stretching to which it is subject it is glistening, desquamates slightly. Instead of appearing as distinct nodules, ions may appear as diffuse infiltrations in the skin which pro- mooth, firm, somewhat elevated and ill-defined plaques. There ssive sebaceous secretion over the tubercles which gives them a appearance, while perspiration is entirely absent. Sensibility is hed over the tubercles, and may be entirely absent. The hair s are gradually destroyed by leprous infiltrations, so that the n them become dry and lusterless and then fall out. The eye- the eyebrows, and the hair of the face fall out in part or totally; s of the eyebrows, particularly in the outer half, is a very char- tic symptom. As the scalp is not usually involved, there is no a in that location.

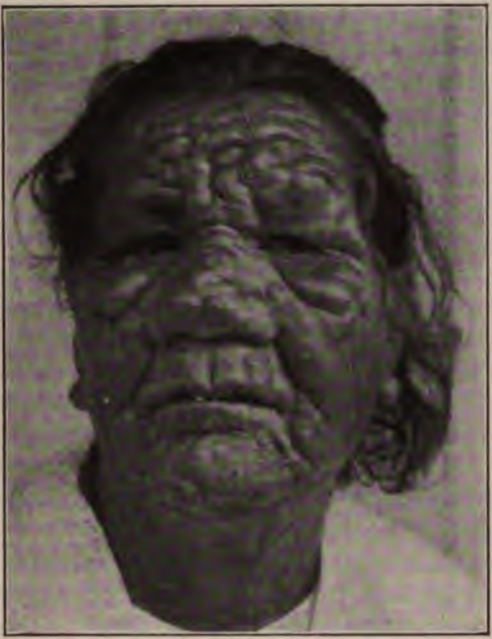

FIG. 185.—TUBERCULAR LEPROSY.

lesions usually appear first and their development is greatest he exposed parts—the face, the backs of the hands, the wrists, and, among people who habitually go barefoot, the dorsa of the feet and the lower parts of the legs. The infiltrations may develop upon any part, but they are uncommon on the back of the neck, the back, the nates, and the flexor surfaces of the limbs, and they are excessively rare on the palms, soles, and scalp. As the lesions develop on the face they produce changes in the features which are characteristic. The nodules develop in particular abundance in the eyebrows, on the forehead, the alæ nasi, the lips, and in the external ears, and as they increase and coalesce the skin of the forehead becomes thickened and nodular with the folds exaggerated, the eyebrows enlarged and prominent, the nose thickened and flat and, as a result of ulceration of the cartilages, perhaps depressed, the cheeks nodular and furrowed, the ears enlarged and roughened and the lobules pendulous. The result on the whole is a great exaggeration of the normal features, producing the characteristic leonine face. With the establishment of the leprous infiltrations upon the face and extremities, indolent enlargement occurs in the cervical, axillary, and inguinal glands.

After tubercles develop they may remain stationary for a time, but with recurrent febrile attacks there are progressive changes in the infiltrations. During these attacks the tubercles may become red, swollen, and tender, and many of them may disappear by absorption, but after each attack the extent of the eruption is increased by the appearance of new lesions. When tubercles disappear by absorption they leave soft cicatrices; when they ulcerate they may slowly heal spontaneously or more rapidly as the result of proper antiseptic treatment, leaving somewhat thickened scars. The ulceration may be accompanied by necrosis of the underlying bone, resulting in the loss of fingers, toes or extremities, or in extensive irregular ulcers. In the flat diffuse infiltrations ulceration may take place at multiple points; the multiple ulcers thus formed may coalesce into irregular, large, indolent ulcers with hard, raised, abrupt borders. Such ulcers may be very extensive, perhaps entirely surrounding an extremity. Along with the ulceration of the infiltrations in the skin, the affected glands break down with the formation of deep ulcers and discharging sinuses.

It is evident that in a persistent, slowly progressing disease with the destructive tendencies of leprosy the possibilities for mutilation are unlimited, and the late stages of severe forms of tubercular leprosy are most pitiful pictures of disease.

Evidences of involvement of the mucous membranes appear along with the first involvement of the skin. According to Hillis, during the febrile attack there appear in the mucous membranes of the roof of the mouth, fauces, and pharynx, red, congested, glazed patches with raised crescentic borders. Later the mucous membranes of the mouth, of the

pharynx and larynx, and of the trachea become dull white. Very early in the disease tubercles develop in the mucous membrane of the nose, especially over the septum, and later, as they ulcerate, there is destruction of the cartilage and of the bone, with sinking down of the bridge of the nose. Infiltration also occurs in the mucous membrane of the tongue, cheeks, hard and soft palate, uvula, tonsils, and larynx. On the tongue the lesions appear as discrete nodules or opalescent spots; ultimately the tongue becomes thickened, raspberrylike, and lobulated. Tu-
ercles also develop in the mucous membranes of the eyes.

As the tubercles increase in number, and later as they ulcerate upon
he mucous membranes, disgusting and serious destructive changes occur.
here is a fetid discharge from the nose, and it becomes deformed from
ceration. Ulceration occurs in the soft palate, pharynx, and larynx;
e patient becomes hoarse, perhaps ultimately speechless; and the breath-
g becomes difficult and wheezing, and bronchial catarrh adds to the
stress. The infiltrations spread to the interior of the eye and sight
ay be lost, either from ulceration of the cornea or from intraocular
owths. Ultimately the lesions may cause the loss of taste, smell, and
ght, and as nerve lesions in the mean time may destroy touch, the
tient may be left with hearing alone.

Sooner or later in tubercular leprosy there is involvement of the
rve trunks with disturbances of sensation similar to those of anæsthetic
prosy. The facial, radial, ulnar, median, and peroneal are the nerves
ually involved. At first there are the painful symptoms that character-
e neuritis; later, after repeated painful attacks and after atrophy of
e nerves has occurred from leprous infiltration, there is anæsthesia.

The average duration of tubercular leprosy is eight to twelve years;
ses may go on for twenty years or more. Occasionally the disease runs
n acute course in which the febrile attacks are severe and frequent,
nd lead to an early fatal termination.

The toxic febrile disturbances which usher in the disease have already
een referred to. These tend to recur with greater or less frequency as
ng as new lesions develop. Their frequency is in proportion to the sever-
y of the disease. In mild cases only two or three such attacks may occur
n the entire course of the disease; in severe cases several attacks may
ccur annually. As the disease progresses cachexia becomes well marked.
ost cases are complicated by nephritis, and a frequent occurrence is
myloid degeneration of the kidneys, liver, spleen, and intestines.
eath may occur from these complications, or the patients may be carried
ff by intercurrent diseases. They are particularly liable to tuberculosis.
he disease in the vast majority of cases slowly but steadily progresses
o a fatal termination. Occasionally, however, it is arrested and recovery
ven may take place.

Anæsthetic Leprosy.—In anæsthetic leprosy there are trophic changes in the muscles and skin and other tissues within the areas supplied by affected nerves, which are the result of the degenerative changes produced by the leprous infiltrations of the nerves. We thus have bullæ, muscular wasting, interstitial absorption of bone, and areas of hyperæsthesia followed by anæsthesia, all of which are attributable to the nerve changes. There is also an eruption of more or less infiltrated erythematous patches, which are, however, probably not the result simply of trophic disturbances; for, as Darier has shown, there is a perivascular infiltration of cells in these patches which is identical in character with that found in the leprous nodules, the difference being only one of degree. Therefore, even in anæsthetic leprosy, speaking accurately, the disease occurs in mixed form, but the symptoms resulting from the nervous changes predominate so largely that the clinical picture is quite distinct.

Anæsthetic leprosy may have prodromal toxic febrile disturbances similar to those of tubercular leprosy. They are usually, however, much less marked, and may be absent or escape notice. In addition to these symptoms there may be prodromal neuralgic symptoms, the result of the early neuritis.

The beginning of anæsthetic leprosy is characterized by the appearance of erythematous patches and of symptoms of neuritis. Early in the course of the disease the ulnar and peroneal nerves are found to be thickened and tender, especially where they pass near bony prominences, and evidences of involvement may at the same time or soon after appear in other nerve trunks. In the areas of distribution of the affected nerves there is at first hyperæsthesia which later, as the nerve undergoes atrophy, is succeeded by anæsthesia. The hyperæsthesia which characterizes the early nerve changes may be so intense that the slightest contact causes pain, and the patient can have relief only by rest in bed. After a shorter or longer time, as the neuritis is succeeded by atrophy of the nerves, anæsthesia takes its place, and this ultimately becomes complete. The areas of hyperæsthesia and anæsthesia may be limited or extend over the greater part of an extremity or the face. These areas may or may not entirely correspond to the erythematous patches.

As result of anæsthesia patients are very likely to suffer from traumatisms which they produce unconsciously, and on account of anæsthesia of the feet the gait becomes uncertain.

There is arrest of secretion of sweat and sebum in the anæsthetic areas, and as the disease progresses the skin becomes dry, inelastic, and parchmentlike.

The other primary symptom of anæsthetic leprosy is the development of erythematous patches from the size of a coin to that of the hand or larger. At first they are reddish and disappear on pressure; later they become pigmented, do not disappear on pressure, and are slightly infiltrated and perhaps elevated. Later in their course there are retrogressive changes which usually begin at the center of the patches; the pig-

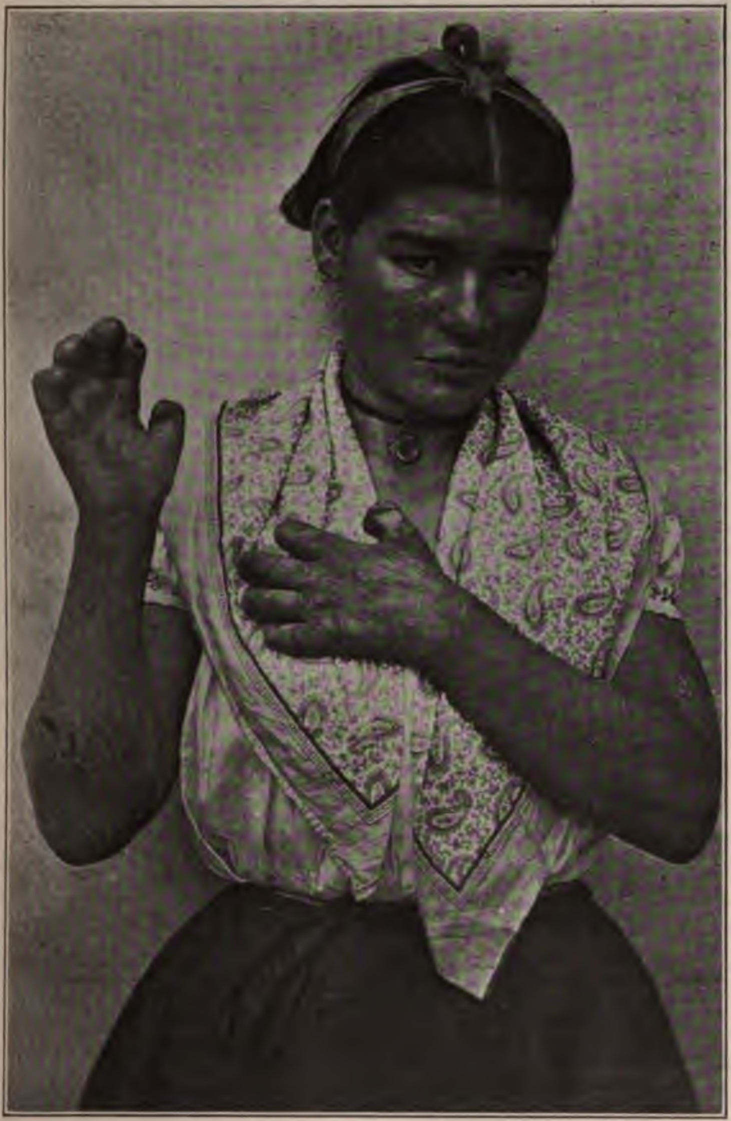

FIG. 186.—ANÆSTHETIC LEPROSY.

ment is absorbed and the skin at the center is left quite anæsthetic and pale or white, or, in some cases, livid. Around these subsiding centers there is usually a border of more active disease, slightly raised, of dark-reddish color, and perhaps studded with vesicles. The patches may remain stationary or they may enlarge peripherally, and then upon the coalescence of adjacent patches circinate figures are formed with a pale anæsthetic center and a dark-red, slightly elevated border. Early in their course there may occur in the patches tingling, or burning, or formication; later they become anæsthetic. In the spreading patch its subsiding center may be entirely anæsthetic while the active border is hyperæsthetic.

The distribution corresponds with that of tubercular leprosy, and, as in tubercular leprosy, the lymphatic glands contiguous to the patches become enlarged.

The bullæ which occur in the areas of distribution of the affected nerves are apparently secondary, the result of the nerve changes. They are of constant occurrence and may appear early in the course of the disease. They vary in size from a pea to a hazelnut, or occasionally much larger. Early in the disease they are usually small and numerous and hyperæsthetic, or of normal sensation; later they are larger, single, and perhaps anæsthetic. They are filled at first with a sticky, yellowish, translucent fluid which in a few days, if they do not rupture, becomes purulent. Rupturing, they leave superficial ulcerated surfaces upon which brownish crusts form. These ulcers may heal quickly, but as a rule persist for weeks or longer. They are followed by slightly depressed white scars of diminished sensation. They may occur upon any part of the body except the scalp. Unlike the macular and tubercular lesions, they are rare on the face and common on the palms and soles. They have been seen on the mucous membranes. Their appearance may continue almost without intermission for several years.

As result of the nerve changes, trophic disturbances sooner or later make their appearance in the muscles supplied by the affected nerves. The first evidence of this may be a decrease in the muscular strength; later there is atrophy. The muscle atrophy is likely to begin in the hands and arms and in the legs and feet, and it ends in characteristic clawlike deformities of these parts. On the feet perforating ulcers of the soles may occur, especially in those who go barefoot. Late in the course of the disease there is further deformity of the feet and hands from caries or interstitial absorption of the bones of the fingers and toes. These may progress until the bones entirely disappear, converting the parts into formless stumps upon which, perhaps, abortive nails persist to indicate the identity of the members.

Upon the face the nerve changes result in paralyses. From paralysis of the orbicularis palpebrarum the eyelids cannot be closed, the lower lids droop, particularly at the inner canthus, and the tears flow down over the cheeks. Complete ectropion may occur, and from inability to cover the eyeball secondary conjunctivitis and ulceration of the cornea occur. From paralysis of the orbicularis oris there is inability to close the mouth, the lower lip droops and the saliva dribbles away. Late in the disease the sense of smell and taste are diminished or lost.

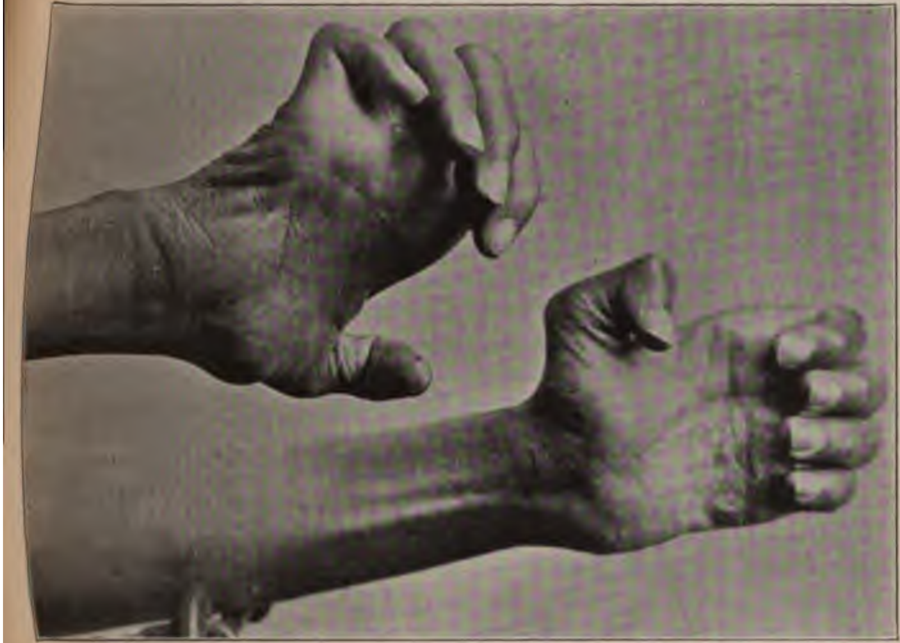

FIG. 187.—CLAW HANDS OF ANÆSTHETIC LEPROSY. (Winfield's collection.)

Either from less active virulence of the organism or from greater resistance on the part of the individual, anæsthetic leprosy pursues a milder and slower course than tubercular. Its average duration is about eighteen years; it may last thirty years or more and the patients may reach old age. If it pursues its course to the stage of extreme paralyses and trophic disturbances which characterize its later course, there is likely to be amyloid degeneration of the kidneys or the development of severe gastrointestinal disturbances which lead to fatal termination. The disease may be arrested, and if this occurs in young persons the erythematous patches disappear and the disturbances of sensation diminish or disappear, so that a complete recovery may take place. Ordinarily, however, when the disease is arrested the anæsthesia and the atrophic changes which have occurred remain.

Etiology.— The bacilli are found in the saliva, the mammary glands and milk, the urine, the semen, and in the secretions from the ulcers. They are found in large numbers in the nasal secretions. It is altogether likely that the disease may be communicated by both direct and indirect contagion. There are undoubted cases of its direct transmission from man to man, and its frequent occurrence in women who wash the clothes of lepers suggests its transmission by clothing as probably a common method. All of the conditions necessary for the spread of the disease, however, are not understood. The activity of the contagion is not great, and there are innumerable instances of families living for years in intimate contact with lepers without any of them becoming leprous. It is rare for physicians or nurses having charge of lepers to contract the disease, although this occasionally happens. The most important factor in infection with leprosy is apparently a residence in a district where leprosy is endemic, and the extreme rarity with which the accident occurs even in leprous localities suggests that individual susceptibility is an important factor.

Leprosy may be contracted at any age. The preponderance of opinion is that it cannot be transmitted by heredity.

Hutchinson has strongly advocated the view that leprosy is transmitted through eating fish. His view is not widely accepted.

Pathology and Bacteriology.—The lepra bacillus resembles the tubercle bacillus very closely; it is, however, straighter, shorter, and sometimes has sharply pointed extremities. Its staining reactions are similar to those of the tubercle bacillus, though it is more easily decolorized by acids. In its late stages of development it shows shining points considered to be endospores. It has a surrounding capsule of glœa, which stains less intensely than the bacillus itself. It has never been cultivated, and inoculations in animals have had negative results.[1] Its existence aside from the disease is unknown.

Leprosy, like syphilis and tuberculosis, is characterized by the production of a particular type of granuloma. The lepra tubercle is composed of connective-tissue cells, many plasma cells, and some mast cells and mononuclear leucocytes, and forms first around the vessels or glandular structures. It is not vascular, and the epithelial structures (glands, etc.) and the fibrous elements are destroyed to a large extent. Scattered throughout the granuloma, within and without the cells, and filling the lymphatic spaces, are enormous numbers of Hansen's bacilli, single and in masses. Peculiar to the infiltration of lepra are the so-called lepra cells, considered as specific by Virchow; these are large, irregular, poorly defined masses, with or without one or more apparent nuclei, and consist principally of bacilli; they are variously considered as mononuclear and giant cells degenerated under the invasion of the bacilli, or as pure masses of bacilli with their surrounding zoöglœa, producing in section an apparent cell form.

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[1] Nicolle, *Annales de l'Institute Pasteur*, has found that monkeys of low type are susceptible to leprosy to about the same degree that they are to syphilis. It may be that the disease is definitely transmissible to anthropoid apes (*Abs. Jour. A. M. A.*, September 22, 1906).

McCoy, *Jour. A. M. A.*, August 22, 1908, p. 690, of the Marine Hospital Service has found a leprosy-like disease in rats in San Francisco. Skin and deep-seated lesions, containing many bacilli, exist in the diseased rats. The bacilli were acid-fast and indistinguishable from the leprosy bacillus. Brinckerhoff thinks that the disease is not identical with human leprosy, but related to it in some way, as bovine tuberculosis is to human tuberculosis.

Clegg, *Philippine Jour. of Sciences*, April, 1909, has succeeded in cultivating the lepra bacillus in symbiosis with the amœba of dysentery.

In the anæsthetic form of the disease there is degeneration of the cutaneous filaments of the peripheral nerves with parenchymatous degeneration and interstitial sclerosis. Bacilli are abundant in the enlarged nerves. They are also found, but sparsely, in the erythematous patches.

Diagnosis.—The picture of even fairly developed leprosy is so characteristic that there is little room for mistake in diagnosis. In doubtful cases of tubercular leprosy the diagnosis may be confirmed by the finding of the bacilli in the nasal secretion or, if this fails, by the excision of tissue from a nodule in which the bacilli are always to be found.

In a doubtful case of anæsthetic leprosy the bacilli may perhaps be found in a piece of tissue taken from the active borders of an erythematous patch. Shepherd has suggested that in cases where the necessity for immediate diagnosis is urgent a piece of tissue for examination should be excised from the enlarged ulnar nerve at the elbow.

The lesions of tubercular leprosy, while presenting a superficial resemblance to other tubercular lesions in the skin, cannot with ordinary care, especially if examination of tissue is made for bacilli, be confused with any of them. Anæsthetic leprosy may be confused with syringomyelia. The presence of the erythematous patches and the palpable enlargement of the ulnar and peroneal nerves in leprosy, along with differences in the character of the nervous symptoms in the two diseases, render a diagnosis upon careful examination not difficult.

Treatment.—The first point in the treatment of leprosy is attention to the general health. Every effort should be made to put the patient's resistance at the highest point. If he is a resident of a leprous district he should if possible be removed. Aside from the aid to be gotten from symptomatic treatment considerable hope may be expected from specific medication. Chaulmoogra oil, used both internally and externally, is the best accredited remedy in leprosy. It is given in doses of from 5 to

75 gtt., t.i.d., in capsules, emulsion, or milk. It is hard to take, and is apt to disagree with the stomach, but tolerance for it may be established and its value is sufficiently well attested to warrant persisting in the effort to administer it. It should also be applied externally to the lesions in the strength of twenty-five per cent in olive oil. As a substitute for it, its active principle, gynocardic acid in the form of gynocardate of sodium or magnesium, has been suggested in doses of 3 to 5 grains in capsules of which ten to twenty are taken daily. Gurjun oil is used in doses of 5 to 10 minims t.i.d. Hoang-nan, dose 3 grains t.i.d. in pill form, is also widely used in leprosy, but is probably not as generally useful as chaulmoogra or gurjun oil. Dyer has had great improvement from the use of hypodermic injections of Calmett's antivenene in doses of 1 to 11 c.c. every day or every other day. Crocker has seen much benefit from the hypodermic injection of corrosive sublimate. Unna recommends for inunction an ointment of chrysarobin and ichthyol (8 parts), salicylic acid (2 parts), and vaseline (100 parts); for use on the face the same ointment, substituting pyrogallic acid for chrysarobin. The use of any sort of greasy application is beneficial.

Lately there has been widespread testimony to the value of x-rays upon the leprous lesions in the skin, and it seems likely that this agent offers far and away our most valuable method of treating the lesions in the skin. There is less reason to hope for benefit from it in the nerve lesions.

The ulcerative lesions of leprosy are to be treated according to ordinary surgical principles with antiseptic applications. Where surgical operations are necessary, as amputations, nerve-stretching, and tracheotomy, they can be successfully carried out in lepers.

XXIII.—RHINOSCLEROMA[1]

Rhinoscleroma is a specific local disease characterized by the development of densely hard, painless, slowly increasing nodules or plaques about the anterior nares and adjacent parts.

Rhinoscleroma was first described by Hebra and Kaposi in 1870.

Symptomatology.—The disease begins with the formation of flat, elevated, sharply defined plates or nodules which are dense, hard but elastic, and painless but tender on pressure. The lesions have the characteristics of a dense, neoplastic infiltration. The surface is of normal color or bright to brownish red, traversed by a few blood vessels, glossy, with smooth or wrinkled epidermis and free from hair and follicles. The surrounding skin is quite normal in appearance.

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[1] Marschalko, "Zur Histologie des Rhinoscleroma," *Archiv*, vol. liii, 1900 (complete study and bibliography).—Wende, *Jour. Cut. and Gen.-Urin. Dis.*, 1896.—Kaposi, p. 533.—Hebra, vol. iv, p. 1.

The growth usually begins upon an ala nasi or the septum nasi. The parts gradually thicken and harden, the alæ are pushed outward giving the appearance of a pug nose, and the parts slowly become rigid and castlike, the nares narrowed and finally occluded. The infiltration of the tissues persistently but slowly extends, involving the lips, mouth, posterior nares, and perhaps adjacent periosteum and bone. Ultimately the deformity may be very great, causing occlusion of the nostrils, stenosis of the mouth, and perhaps of the larynx. According to Kaposi, whose experience with the disease almost equals that of all other observers, the lesions never ulcerate, and even wounds left from excision of portions of the tumor do not suppurate, but are recovered with epidermis. According to other observers, however, there may be in a late stage of the disease superficial excoriations with a viscid exudate which dries into yellow crusts, and superficial ulceration may occur upon mucous surfaces. Frank ulceration has been observed in cases of Zeissl's and Lang's.

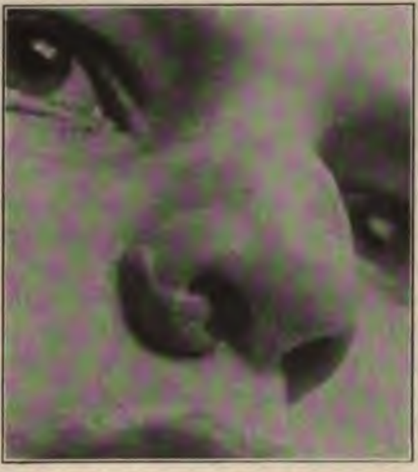

FIG. 188. — RHINOSCLEROMA, BEGINNING CASE. (Grover W. Wende's collection.)

The nose is the usual initial site of the disease whence it spreads to the adjoining structures. It has been seen in the external auditory canal, the ear, and the trachea.

The course of the disease is slow but progressive, and it may continue for many years. It always, however, remains local. Spontaneous involution does not occur, and even after complete removal it usually recurs.

There is no spontaneous pain in the lesions, but they are painful on pressure. The general health is unaffected except indirectly from the obstructions in the mouth, nose, or larynx. These, when the condition becomes extensive, may cause very distressing symptoms or death.

Etiology and Pathology.—The disease is very rare, and most of the cases have been seen in Austria and southwest Russia. A very few cases in foreign-born patients have been seen in America by Jackson, Klotz, Allen, and others. Wende has demonstrated one case in a vigorous boy always a resident of Buffalo, N. Y., and of well-to-do American parents. It occurs in both sexes, in persons of all classes, usually between the ages of fifteen and forty, and in patients who present no definite dyscrasia.

The only appreciable change in the epidermis is an increase in the interpapillary projections, which form branched prolongations downward into the corium. The papillæ of the corium are correspondingly elongated. The corium is filled with a dense, cellular infiltration consisting principally of young connective-tissue cells and plasma cells. Certain large, round cells with several nuclei occurring in this infiltration have been described as hyaline and colloid cells, but have been shown by Marschalko to be degenerated plasma cells. Peculiar to this disease are the cells described by Mikulicz, which are connective-tissue cells containing masses of the bacilli in zoöglœa. Surrounding .the cellular infiltration is a very hard and dense white fibrous tissue.

The bacillus of rhinoscleroma was first described by v. Frisch in 1882; its pathogenicity for animals was demonstrated by Pawlowsky in 1890; and it is now generally accepted as the specific etiological agent. It is a rod-shaped bacillus, 2 microns long by .5 micron in thickness, with a distinct capsule, and a central nonstaining area. It grows on all ordinary media with formation of thick masses of zoöglœa. In the tissues the bacilli are to be found both in the cells and free, in groups and masses. They occur both in the immediate lesions and in the related lymphatic glands. They are best stained in the tissues by Gram's method.

Diagnosis.—The characteristic features of the disease are its location, its progressive, painless character, the smooth ivory hardness of the lesions, and the absence of softening and ulceration.

The lesions most resemble syphilitic gummata, from which they differ in nearly all the characteristic features enumerated above, and by the entire lack of influence of antisyphilitic treatment. Confusion is also possible with keloid, rhinophyma, and epithelioma. The histological structure and the presence of the specific organism differentiate the disease from both keloid and epithelioma. Further, keloid follows injury, is sometimes spontaneously painful, and has the density of connective tissue. Epithelioma is indurated, but not of the dense hardness of rhinoscleroma. In its border can be found characteristic waxy nodules, and it tends to destructive ulceration. In rhinophyma, which superficially resembles rhinoscleroma, the tissues are soft, exuberant, and cauliflowerlike, and pitted by greatly distended, gaping sebaceous follicles.

Treatment.—The treatment is unsatisfactory. Destructive measures, including excision, are usually followed by recurrence, but may be necessary in order to preserve the patency of the mouth or nose. Lang has had comparatively good results from injecting into the tumors solution of salicylic acid (one per cent) and of sodium salicylate (two per cent). Theoretically, x-rays offer prospect of benefit.

XXIV.—GANGOSA

Synonyms: *Rhinopharyngitis mutilans* (Leys). *Ogo* (Chamorro).

"Gangosa, a Spanish word meaning muffled voice, is the name employed by the Spaniards in the Ladrone and Caroline Islands to describe a disease characterized by a destructive ulceration, usually beginning on the soft palate, pillars or uvula, and extending by continuity to the hard palate and nasal cavity, larynx, and even to the face. Active ulceration is followed after a variable period by cicatrization or chronic ulceration. Mutilation always results. Constitutional symptoms are either slight or absent." (Mink and McLean.)

Gangosa begins as a superficial ulcerative process, usually in the soft parts of the pharynx or nose. The ulceration extends with greater or less rapidity, attacking bone as well as soft parts. Periods of activity are followed by periods of quiescence, during which time scar tissue formation occurs, and thus in the course of time great deformity of the affected parts takes place. The disease is usually very chronic, lasting for years. It remains localized, does not cause constitutional symptoms, and when death occurs it is from intercurrent diseases to which cachexia makes the victims more susceptible.

There is a fulminating type which occurs in young children in gangosa families. With rapidly developing throat symptoms and extreme toxemia, the whole picture closely resembles diphtheria. Most of these cases prove rapidly fatal. The acute type is said to be distinguished from diphtheria by the absence of the Klebs-Loeffler bacillus.

Gangosa is very widely distributed in Oriental and tropical countries. It occurs in the Philippines, Pacific, and Indian Islands, in the West Indies and South America. It is a disease associated with poverty and squalor, but apparently no race is exempt. Stitt has found a case in a United States marine who lived in intimate relations with gangosa families in Guam. It attacks all ages, but is commonest during adolescence and early adult life. Attention has been called to the disease and our knowledge of it much increased by men in the medical services of the United States who have been working in our new colonies; notably by Mink and McLean, Stitt, Musgrave and Marshall, and Geiger. Fordyce has carefully studied and recorded one case in New York City in a negro from Panama. The lesions are histologically infectious granulomata, probably due to a specific organism which has not been definitely determined. Geiger has found an organism closely resembling the diphtheria bacillus in all the cases examined by him; he has found it in pure cultures in the conjunctival sac in the cases where the eyes were involved. Its histology and clinical course distinguishes it definitely from tuberculosis and syphilis, which it most closely resembles, as well as from blastomycosis, actinomycosis, rhinoscleroma, and leprosy. The incubation of the disease is unknown; it is apparently several months. There is no evidence that infection occurs by direct inoculation, although all the facts indicate that the disease is contagious; somewhat more so than tuberculosis, and more so than leprosy. Unhygienic living is the most important factor, after infection, in its etiology.

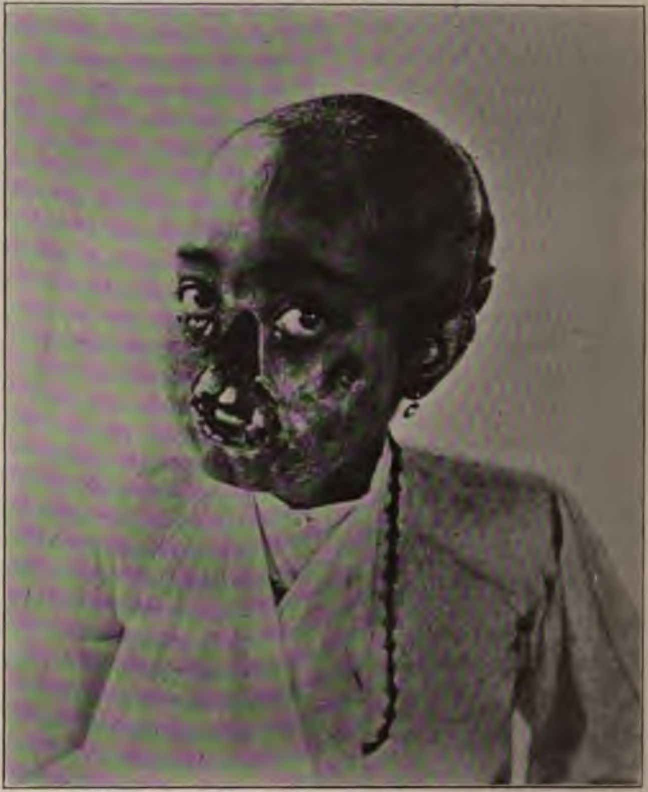

FIG. 189.—GANGOSA. (Mink and McLean.)

The treatment, except in so far as general tonic measures are indicated for overcoming cachexia, is local and has for its aim the destruction of infected areas. Tincture of iodin freely applied is highly recommended for this purpose. For large masses of diseased tissue, strong caustics, even the actual cautery, may be indicated. Antiseptic mouth washes like potassium permanganate (one per cent) are useful where ulceration is abundant. Specific anti-syphilitic treatment is not effective.

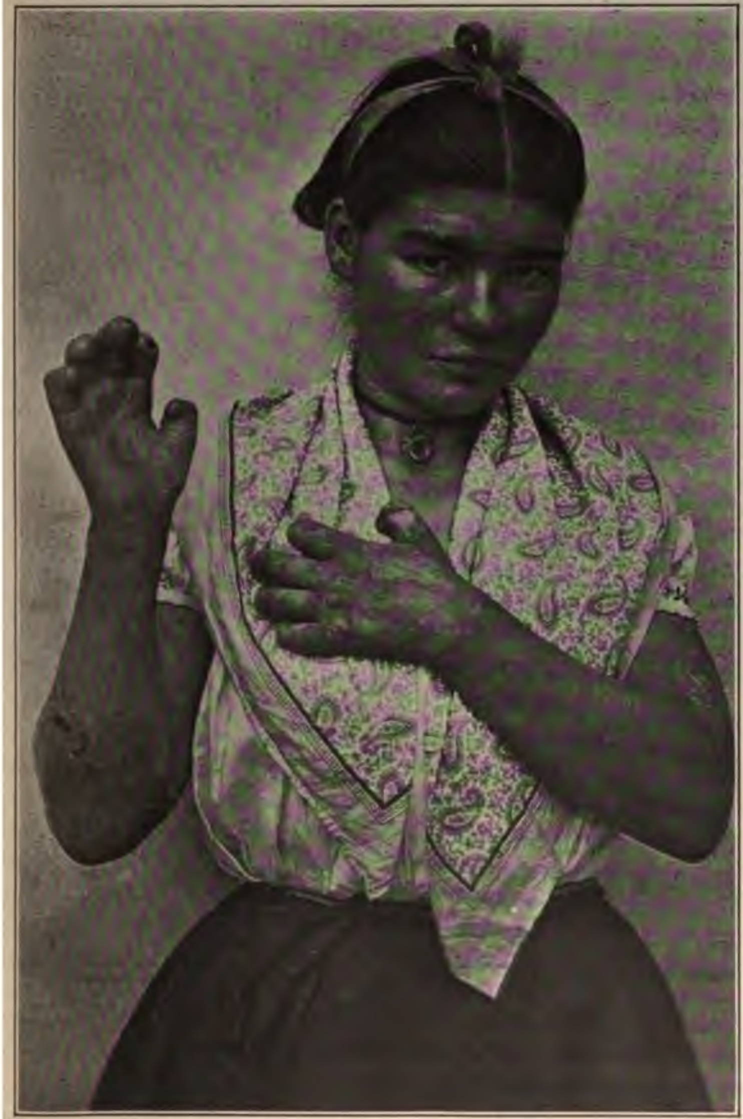

FIG. 186.—ANÆSTHETIC LEPROSY.

rythematous patches from the size of a coin to that of the hand or er. At first they are reddish and disappear on pressure; later they me pigmented, do not disappear on pressure, and are slightly infil- ed and perhaps elevated. Later in their course there are retrogres- changes which usually begin at the center of the patches; the pig-abscesses, from the pus in which pure cultures of the organism can usually be obtained.

The recognition of blastomycetic dermatitis as a pathological entity dates from the meeting of the American Dermatological Association in May, 1894, when Gilchrist demonstrated sections containing budding organisms which had been taken from a scrofulodermalike lesion. In November, 1894, Busse reported a fatal case of pyæmia with subcutaneous abscesses and cutaneous lesions in which the pathogenic agent was a yeast. July 7, 1894, Buschke (Greifswald Medical Society) referred to a case with skin lesions in Busse's clinic in which he had found coccidial bodies. Buschke and Busse first demonstrated the significance of the organism and established that it was a saccharomycetic fungus and reproduced the disease by inoculation on the skin of their patient, although Gilchrist in his first

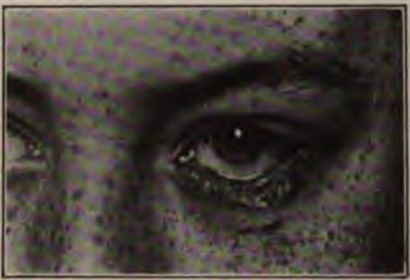

Fig. 190.—Blastomycosis of Lower Lid, a Common Beginning Point, Girl Fourteen. (Author's collection.)

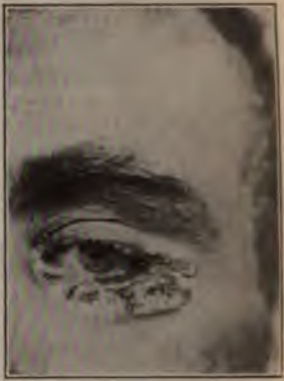

Fig. 190a.—Blastomycosis of Lower Lid, showing Beginning Characteristic Ectropion. (Author's collection.)

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vol. ii, 1902, p. 1321.—Second Annual Report of the Cancer Com. to the Surgical Dept. of Harvard Medical School, *Jour. Med. Resch.*, vol. vii, 1902, No. 3, "Researches on blastomycetes, protozoa, cancer bodies, and cell inclusions."—Buschke, *Bibliotheca Medica*, B. ii, h. 10, 1902 (complete monograph).—Brayton, *Jour. A. M. A.*, February 1, 1902.—Shepherd, *Jour. Cut. and Gen.-Urin. Dis.*, 1902.—Dyer, *Amer. Medicine*, October 25, 1902.—Fischkin, *Chic. Med. Recorder*, 1903, p. 408.—Varney, H. R., *Detroit Med. Jour.*, 1903–4.—Montgomery, *Jour. Cut. Dis.*, 1903, p. 19.—McCarrison, *Indian Med. Gaz.*, April, 1903.—Pusey, *Jour. Cut. Dis.*, 1903.—Evans, *Jour. A. M. A.*, 1903, p. 1772, "A Case of Cutaneous Blastomycosis from Accidental Inoculation."—Squeira, *Brit. Jour. Derm.*, vol. xv, 1903, p. 121.—Ormsby and Miller, *Jour. Cut. Dis.*, 1903 (systemic case).—Otis and Evans, *Jour. A. M. A.*, October 31, 1903.—Unna, *Münch. med. Wochensch.*, 1904.—Dubreuilh, *Jour. de méd. de Bordeaux*, 1904.—Wright, *Northwest Lancet*, 1904.—Löwenbach and Oppenheim, *Archiv*, 1904, vol. lxix, p. 121.—Gilchrist, *Jour. Cut. Dis.*, 1904, vol. xxii, p. 107.—Cleary, *Trans. Chic. Path. Soc.*, May 1904 (systemic case).—Ricketts, *ibid.*, May, 1904.—Eisendrath and Ormsby, *Jour. A. M. A.*, October 7, 1905 (systemic case).

ation held, without having fully demonstrated it, that the is were due to the fungus. Since that time numerous cases observed. It has for some inexplicable reason, proved by frequent in Chicago, and for our present knowledge of the are especially indebted—after Gilchrist—to the thorough the disease made in Chicago by Ricketts, and Hyde and ry and Ormsby.

me blastomycetic dermatitis was applied by Gilchrist; in view r findings that the disease becomes systemic at times, the name sis suggested by Hyde is perhaps better.

omatology.—Blastomycosis of the skin begins as an indolent, ory papule or papulo-pustule upon the tip of which there de- vartlike hypertrophy. As the lesions become mature there is he papillæ more or less exudation of pus which dries into a ust. Such a lesion be larger than a a, but it presents all teristics of the larg- es. It is indolent tically painless, its r somewhat rounded hened with warty, ojections. The base d upon squeezing it f pus may be made between the papillæ. r of the lesion slopes nd is of purplish or inflammatory red in it may be seen naked eye, or better and glass, pinpoint- w points which rep- ute abscesses deep in mis. These minute abscesses from which res of the organism btained are a char- feature of the lesions. r lesions are exagger- these small lesions hange of the essential features. The small lesions increase peripheral growth and by the coalescence of adjacent le-

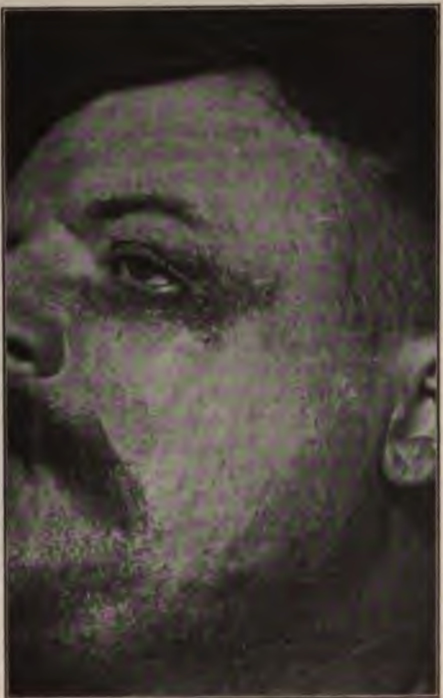


FIG. 191.—BLASTOMYCOSIS SHOWING ACTIVE LESIONS ABOUT THE LOWER LID AND SCARRING OF THE ENTIRE CHEEK FROM THE DISEASE. (Author's collection.)

sions. In this way lesions may be formed from the size of a coin to that of the hand, or, in extreme cases, much larger. The large patches may be elevated to a height of three eighths of an inch to an inch. They present a papillary, wartlike surface with exudation between the papillæ of an abundance of pus and more or less crusting upon the surface. Upon removal of the crusts there are usually exposed suppurating papillary growths which bleed easily, or perhaps papillary growths which are covered with horny epidermis, but from the interstices between which there is free exudation of pus.

The patches may not show any definite ulceration, indeed they may remain covered with thickened warty epidermis without exudation of pus except upon pressure, but as a rule ulceration takes place sooner or later. In such cases there will be a center with a single or several ulcers surrounded by a periphery of the characteristic warty growths; or ulceration will occur on the concave side while the characteristic papillary growths spread on the convex side. The ulcers may heal spontaneously, and thus the formation of scar tissue will add another feature to the appearance of the lesions. In some cases entire patches disappear spontaneously, and the disease may again develop in the scars. Ordinarily the scars which form are soft, smooth, sharply defined, and not thickened; at times there is very little scarring. Occasionally the scars are rather thick, causing a good deal of contraction and, as in my case involving the entire face, illustrated herewith, may

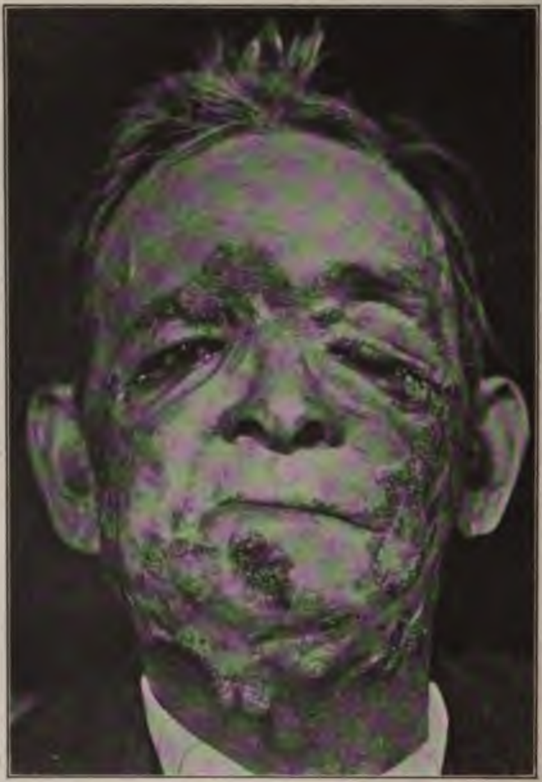
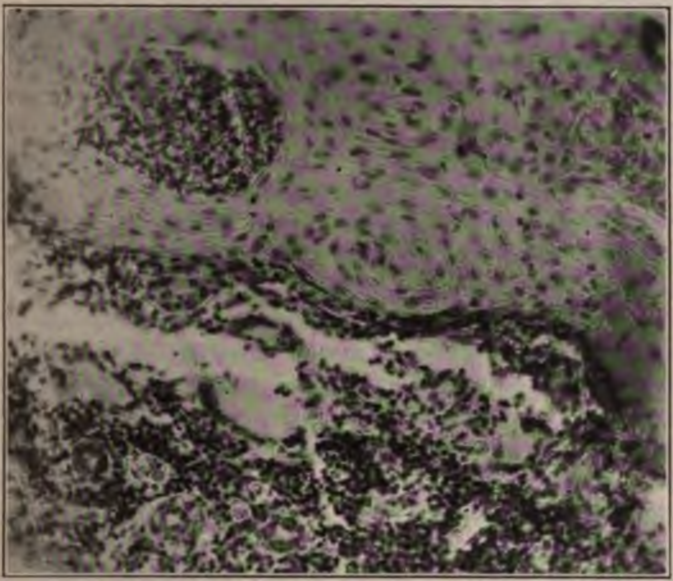


FIG. 191*a*.—BLASTOMYCOSIS. In addition to the involvement of the face there were many lesions on the scalp and on both hands and arms. Duration more than twenty years. (Author's collection.)

duce thick, disfiguring scars. When the lesions occur upon the ver lid, as is frequent, ectropion as a result of destruction of the and contraction of the scar is a characteristic feature of the picre.

The disease may develop upon any part of the body. It has occurred imarily on the legs, thighs, knees, buttocks, and other covered parts, t it occurs most frequently upon the hands and face. The lower lid l the adjacent skin is a favorite site for its development. The disease



192.—BLASTOMYCOSIS. Note epithelial hyperplasia and inter-epithelial abscess in epidermis, and giant cells and blastomycetes in corium. × 200. (Author's collection.)

present itself in only one lesion, but after it has persisted for a ger or shorter time other lesions are apt to occur. These may be on ant parts of the body, and there is little doubt that they are proed by autoinoculation. The glands adjacent to the lesions do not me involved in the disease except in systemic cases, although they , of course, become enlarged from secondary infection.

The disease is exceedingly chronic. Its spread is slow, and spontaus involution may occur. On the other hand, it shows a persistent lency to relapses, so that even after apparent cure one may be disointed by its return. The entire course of the disease may extend r many years. In one of my cases it had probably existed for more n twenty years.

The lesions are painless and only slightly tender on pressure. So long as the disease remains confined to the skin there is no constitutional disturbance. It was first thought that the disease was always local, but systemic involvement has now been demonstrated in several cases. In one case the systemic invasion developed seven years after the cutaneous lesions, in two others it preceded the cutaneous lesions. In such cases the organisms are found *post mortem* in the blood and in the viscera.[1]

Etiology and Pathology.—The disease is due to infection with blastomycetes. It has been reproduced repeatedly in guinea pigs and rabbits by inoculations with pure cultures. In Evans's case a lesion developed a week after an injury on the finger at an autopsy upon a case of blastomycosis. Presumably infection occurs only through injuries.

Both sexes are attacked by the disease with about equal frequency. It is usually seen in adult life, but Kessler has reported a case in a child eight months old. All of my cases have been in the working classes, but they have not been by any means in the lowest class.

The disease is rare; to this statement must be added, except in Chicago. In Chicago so many cases have been seen within the last few years that the disease is no longer a clinical curiosity. No association has been established with any definite etiological factors. Most of the cases have developed spontaneously without any history of association with other cases. I have had two cases from one small village, but in patients who were in no way associated.

Histology.—The characteristic histological features of the cutaneous lesions of blastomycosis are proliferation of the epithelial cells of the rete, formation of miliary abscesses within the epidermis, and acute to chronic inflammatory changes in the corium.

The stratum corneum is in some places broken through or lacking, in others thickened as a result of premature keratinization, which results occasionally also in the formation of pearls within the epithelium. The cells of the rete are large, swollen, with prickles very apparent, and within and between the cells are polynuclear leucocytes. The hyperplasia of the rete results in the formation of long irregular downgrowths into the corium, which resemble greatly in appearance squamous-celled carcinoma, and within which are located miliary abscesses of size varying from those containing only a few leucocytes to those visible to the naked eye and extending to the surface or to the corium. These foci contain polymorphonuclear leucocytes, cellular detritus, sometimes giant cells similar to those of tuberculous tissue, and varying numbers of the specific organisms. The basal layer of cells is intact and continuous. The appendages of the skin disappear early in the course of the lesions.

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[1] Bassoe (*Trans. Chic. Path. Soc.*, December, 1905; *Jour. Infect. Dis.*, 1906, vol. iii) has reported a case of systemic blastomycosis with death without cutaneous lesions, there being blastomycosis of various internal tissues, including vertebræ and cord. Christensen and Hektoen (*Jour. Amer. Med. Assoc.*, July 28, 1906) have also reported two cases of generalized blastomycosis, both of which began with acute febrile symptoms, followed in the course of a few days by lesions of blastomycosis in the skin.

In the corium the changes are those of an inflammatory process of more or less severe type. The vessels are dilated and their walls show more or less hyperplasia. There is cellular infiltration throughout the corium, consisting of polymorphonuclear leucocytes, and young connective-tissue cells, and occasionally there is a focus of more dense infiltration with one or more giant cells and one or more of the organisms of the disease.

Bacteriology.—The organisms as seen in the tissues occur most frequently in the miliary abscesses, and occasionally in the corium. They are rounded or oval, on an average twelve microns in diameter, and possess a distinct capsule bordered by a narrow clear zone, within which is a finely granular protoplasm. They occur singly or in pairs, and budding forms may be observed.

The proper classification of the organisms found in blastomycosis is as yet uncertain. Cultures vary greatly in form and appearance of growth in different cases, and indeed from the same cases, and there is much variation in cultures of the same organism on different media. It seems most probable that there are several of the yeast and mold fungi which are pathogenic and may produce lesions of great clinical similarity. Ricketts has attempted a classification under the genus Oïdium, with the subfamilies Blastomyces, Oïdia, and Hyphomyces; but this has not as yet been generally accepted.

In the tissues the organisms appear as rounded capsulated bodies, with granular and sometimes vacuolated protoplasm, varying in size from one to three times the diameter of an erythrocyte. They reproduce within the body by gemmation without sporulation, differing in this respect from the organisms found in the cases reported by Montgomery, Ophüls, and others from the Pacific coast, and named by them Dermatitis Coccidioides, the peculiarities of which are absence of budding and multiplication by endosporulation within the tissues. Cultures may be obtained on any of the ordinary laboratory media, best on maltose or glucose agar, by inoculation with the pus from one of the minute abscesses. Growth develops in from two to eight days on glucose agar, being white and fluffy with aërial hyphæ, while on glycerine agar it is more pasty, forming folds and depressions. The growth consists of budding organisms or of fine segmented branching mycelia which may possess lateral conidia. In the mycelia are to be seen refractile bodies, probably spores, and among the branching fibrils are round, double-contoured bodies filled with refractile granules—evidently spore capsules with spores.

Diagnosis.—The clinical picture of blastomycosis is definite. The most characteristic feature is the occurrence of the minute, deep-seated abscesses in the border, from which the organism can readily be obtained. The diseases with which it may be confused are tuberculosis verrucosa cutis (the disease which it most closely resembles), vegetating syphilide, and epithelioma. The characteristic clinical features are sufficient to differentiate it readily from any of these diseases, but the diagnosis always needs confirmation by demonstration of the organism.

The organism can readily be demonstrated by placing a smear of pus or a bit of teased tissue on a slide, covering it with twenty- to thirty-per-cent solution of potassium hydrate and pressing down a cover glass upon it. After ten minutes to an hour the tissue and pus are disintegrated by the caustic potash, while the budding organisms are resistant to it and can readily be demonstrated. Cultures should also be made in doubtful cases.

Prognosis.—The prognosis in cutaneous blastomycosis is good if proper treatment can be persistently carried out. Unless the disease is completely eradicated it is very prone to recur; recurrence may take place in apparently healthy scars. The systemic cases are fatal.

Treatment.—The local treatment of blastomycosis consists in measures directed to the destruction of the diseased tissues. This was formerly done by curetting or excision. Excision, if complete, is usually successful. These procedures have been largely replaced, at least in Chicago, by the local use of x-rays combined with the internal administration of potassium iodide. The application of x-rays in these cases usually has to be carried to the point of producing dermatitis, and in two cases in my experience the lesions have yielded only after the most persistent treatment. While under treatment the lesions should be cleaned with antiseptics and have dry or, preferably, wet antiseptic dressings.

From the favorable effect of potassium iodide in actinomycosis Bevan suggested its administration in blastomycosis, and combined with the local use of x-rays it has proved a satisfactory method of treatment. Moderate doses of potassium iodide may be sufficient, but in some cases 300 to 500 grains daily have been necessary. Occasionally potassium iodide alone will cause the lesions to disappear entirely. Usually it causes great improvement, but not entire disappearance of the lesions, and to get complete results the local irritation produced by x-rays or destruction by curetting must be added. The use of copper sulphate considered in connection with actinomycosis is worthy of trial, especially in the systemic cases, for in these potassium iodide has proved ineffective.

PROTOZOIC DERMATITIS, DERMATITIS COCCIDIOIDES, COCCIDIOIDAL BLASTOMYCOSIS [1]

Closely allied to blastomycosis, if not identical with it, is the so-called protozoic dermatitis described by Wernicke, of Buenos Ayres, in 1890, and by Rixford and Gilchrist in 1896. Two cases have been reported in Buenos Ayres and five in the United States, all the latter in men who ad lived in adjacent valleys in California. I have had one case in a oreign-born woman in Chicago. D. W. Montgomery and H. Morrow, of an Francisco, who have studied the disease very carefully, regard it as fferent from blastomycosis. The skin lesions are similar, but the sease differs from blastomycosis in that in most cases it has developed the skin after deeper involvement, and in that all of the cases have en fatal.

The bacteriology of the affection as compared with that of blasto-ycosis has been referred to under the bacteriology of blastomycosis.

In the discussion of Montgomery's paper before the American Der-atological Association in 1904, when he presented his reasons for re- rding blastomycetic dermatitis and dermatitis coccidioides as separate seases, Gilchrist and others agreed that neither the clinical picture nor he bacteriological findings in the cases were distinctive enough to differ-tiate the disease from blastomycosis.

HYPHOMYCETIC DERMATITIS [2]

Another condition closely allied to blastomycetic dermatitis is the yphomycetic disease of Duhring and Hartzell, the lesions resembling hose of lupus verrucosus. From abscesses in them they obtained mycelia nd spores.

SPOROTRICHOSIS

Sporotrichosis is an infective granuloma involving the skin and sub-utaneous tissue, and less frequently other structures, produced by a pecific fungus, the sporothrix schenckii. As it occurs in the subcuta-eous tissues it closely resembles, clinically, chronic glanders. The first case was described by Schenck in 1898; two years later Hektoen and Perkins reported a second case with identical microbic findings and named the organism, whose pathogenicity both reporters had established, sporothrix schenckii. In the meantime Brayton had reported, without microbic examination, another case identical in its clinical features with the other two. De Beurmann and Raymond in 1903 reported a case, and since that time cases have been reported in Paris and in various parts of the world.

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[1] Wernicke, R., *J. de Micros.*, Paris, 1891, xv: *Centralbl. f. Bakt. u. Parasit.*, 92, Bd. xii.—Rixford and Gilchrist, *Johns Hopkins Hosp. Reports*, vol. i, 1896.—osados, A., "Psorospermiosis Infectante Generalizada," Buenos Ayres, 1897–98.—ontgomery, D. W., *Brit. Jour. Derm.*, No. 144, vol. xii, 1900 (with bibliography).—phüls, *Phila. Med. Jour.*, 1900.—Seeber, G. R., *Thesis*, University of Buenos Ayres, 00.—Montgomery, Ryfkogel and Morrow, *Jour. Cut. Dis.*, vol. xxi, 1903.—Mont-omery and Morrow, *Jour. Cut. Dis.*, vol. xxii, 1904.—Montgomery, D. W., *ibid.*, arch, 1905.—Wolbach, J., *ibid.*, January, 1905.

[2] *Amer. Jour. Med. Sci.*, March, 1895.

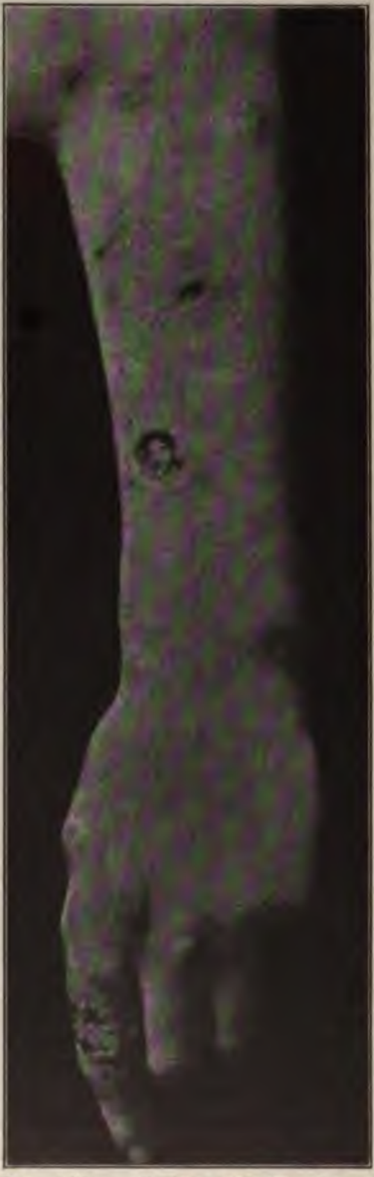

FIG. 193. — SPOROTRICHOSIS, LATE STAGE, SHOWING ORIGINAL LESIONS ON INDEX FINGER, NODULES AND SCARS. (Hektoen and Perkins.)

In each of the first three cases there was a history of a wound of a finger followed by a low-grade infection which invaded the lymph channels and after several weeks produced subcutaneous nodular swellings in the forearm that finally suppurated with the production, when the abscesses opened upon the surface—not an invariable occurrence—indolent sinuses or deep ulcers. The cases were intractable and extended over three to six months before healing occurred. These cases typify the disease as it has generally been observed. The indurated nodular lesions in the skin develop slowly in the subcutaneous tissue; in the course of four to six weeks they become painless "cold" abscesses, which show little tendency to enlarge, and may or may not break down. These lesions heal with difficulty and leave deep, thick scars. They contain a gelatinous pus from which pure cultures of the sporothrix are obtainable. The hands are the most frequent site of infection, but various parts of the body have been involved: Foot, Greco's case; eyelids, Danlos and Bland's case. From one location it may develop over many parts f the body (Letulle's case). De Beurmann and Gougerot and Letulle ave found the lesions on the mucous membranes of the mouth, in association with skin lesions, and this is interesting in connection with he fact that the organism has been found as a saprophite in the mouths of men and rats. Lutz and Splendore have shown that rats may have he disease in the tail and feet. Letulle's case showed extensive indoent ulceration of the buccal and pharyngeal mucosa, resulting in a ysphagia which caused the patient's death. In a case of Massary, oury, and Monier-Vinard the disease was localized in the triceps, and ow de Beurmann, Gougerot, and Vaucher have demonstrated the ocurrence of a sporotrichial periostitis and of sporotrichial osteomyelitis, ith a predilection for the tibia. These lesions are indistinguishable clinally from those of syphilis and tuberculosis. Systemic sporotrichosis s not been found. The cases have shown no constitutional symptoms.

Although few cases have been recognized, the wide distribution of the ses reported—Baltimore and Chicago, California, Kansas, N. Dakota, rance, Italy, Brazil and Argentina—indicate that the disease is not re. Cases in the past have probably usually been considered tuberlosis or syphilis. Histologically, the lesions are indistinguishable from berculosis. Excepting a case reported by Dor, in which the spororix had distinguishing characteristics, the organism in all the cases apparently identical. It may be found as characteristic ovoid bodies n pus and in the tissues in polynuclear and giant cells, but this is ery difficult, and cultures are usually necessary. It may be grown eadily at room temperature on most media, the French preferring Saouraud's medium for the tricophyton fungi. Growths develop in four r five days. It is a strict aërobe. On solid media it produces an paque, grayish white or white growth with a radiating fringe of mycelia nd extends down into the media as a white, coherent mass; later the ass becomes dark brown or black. It liquefies gelatine slowly. In quid media it grows in tufts and balls. The fungus consists of branchg, septate mycelia, upon which ovoid bodies (spores) develop undantly by budding upon the extremities and sides of the filaments. he mycelia and spores stain readily with ordinary stains and by Gram's thod. Widal and Weil have shown that the spores agglutinate when xed with the patient's serum in dilutions of 1-200 to 1-1500; and y have described a complement fixation test for the disease. Rats d mice are readily inoculated with the organism; dogs and guinea pigs less susceptible.

Potassium iodide is specific in the diseases. It should be given in es of 10 to 30 grs. t.i.d. Locally, a dressing should be used, wet with following solution: Iodin, 1 gm.; K I, 10 gms.; water, 500 cc. der this treatment healing occurs in four to six weeks.

II.—ACTINOMYCOSIS[1]

SYNONYM: *Lumpy Jaw.*

Actinomycosis is a specific infectious disease caused by the ray fungus and characterized by the development of indolent granulomatous tumors which tend to break down with a discharge of sero-purulent fluid containing characteristic yellow granules made up of the fungi. It is a relatively rare human disease.

All tissues and probably all organs may be affected by actinomycosis. The disease primarily develops at the point of infection and tends to remain localized in this situation, although metastatic lesions and general infection are ultimately likely to occur. According to Leith's statistics of 430 cases the original site of the disease was as follows: head and neck, 52 per cent; tongue, 3.7 per cent; abdomen, 21.6 per cent; lungs, 13.2 per cent; skin, 2.5 per cent; doubtful, 6.9 per cent. It is evident from the above statistics that primary actinomycosis of the skin is exceedingly rare. When the skin is involved it is usually by the spread of the disease from underlying tissues.

There are no characteristic clinical symptoms by which actinomycosis in any organ can be detected. It begins with the formation of a tumor or tumors usually indolent, which, as far as their clinical characteristics go, might be carcinomata, sarcomata, tubercular or syphilitic deposits, or chronic abscesses. It is only when the lesions break down and the characteristic granules are found that the character of the disease definitely declares itself.

Symptomatology.—The incubation period of actinomycosis is variable and may be very long; weeks, months, or years (Müller's case, two years) may pass after infection before a tumor develops. The disease begins in the skin as a subcutaneous nodule or group of nodules over which, as the surface becomes involved, the skin is tense and glossy and of red or purplish color. The nodules vary in size from a pea to a walnut. Sooner or later the centers disintegrate, they become fluctuating, and the skin over them breaks down at one or more points, from which there exudes a sero-purulent discharge more or less mixed with blood. The lesions usually occur in groups. Ordinarily this is an irregular group with piling up of the lesions at the center, but the grouping may be in circles or in lines. As the lesions increase in number they coalesce and form swollen, glossy, purplish-red nodular patches ous unhealthy sinuses opening upon them and with spots granulations exposed. Sometimes the ulcers become funhe formation of large, purplish, vegetating, papillomatous ıe indolent tumors of s resemble most closens of scrofuloderma merous sinuses which ı with underlying foci sis.

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[1] Wallhauser, *Jour. Cut. Dis.*, 1904.—Crookshank, "Text-book of Bacteriology and Infective Diseases," 1896.—Ackland, Article "Actinomycosis," Allbutt's "System of Medicine."—Pringle, *London Med.-Chir. Soc. Trans.*, 1894.—Sawyer, *Jour. A. M. A.*, May 11, 1901.—Ewing, *Johns Hopkins Hosp. Bull.*, November, 1902.—Howard, *Jour. Med. Resch.*, 1903, vol. ix.—Stokes, *Amer. Jour. Med. Sci.*, November, 1904.

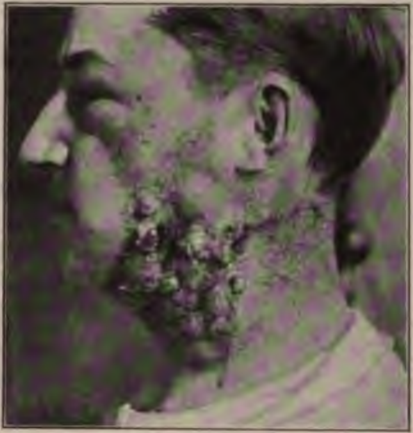

FIG. 194.—ACTINOMYCOSIS.
(H. P. Wells's photograph.)

ıre cases where infecprimarily through the sease probably begins ımmatory vesicle or ard) or as an inflamule or tubercle which evelops into the charcerative nodule.

ıe nodules of actinoeak down there are pus, usually in abunharacteristic granules of the fungus. These are sulphur yelwhen seen by reflected light; when the pus is collected in a d then examined by transmitted light they appear as brownish-brown granules embedded in mucopurulent material.

al location of actinomycosis of the skin is about the face specially in the tissues just under the jaw. Rarely the skin or abdominal wall becomes involved by spread of the disease eper structures. The lesions about the face and neck usually n infections in the mouth. Here the disease begins as an rocess upon the surface of the jaw and spreads downward maxillary region. Metastasis of the disease through the lymare, perhaps due to the relatively large size of the fungus compared with the lymphatics, and lymphatic enlargement e place ordinarily except as result of secondary infection.

ycosis of the tongue is well recognized in cattle in the condias "wooden tongue." There form in the body of the tongue ıodules which break down and form chronic ulcers. The lts frequently in an interstitial myositis. There is atrophy ular fibers, and from the formation of new connective tissue becomes hard and boardlike. This form of the disease has re, been recognized in man.

elopment of the lesions of actinomycosis is slow and the disrsist indefinitely.

The lesions of actinomycosis in the skin are practically painless. They may, however, be associated with extreme pain as result of the presence of lesions in the temporo-maxillary region. Constitutional symptoms are absent in actinomycosis of the skin, unless they result from lesions involving other structures, or from the occurrence of pyæmia.

Etiology and Pathology.—Actinomycosis has repeatedly been produced in guinea pigs, rabbits, and calves by inoculation of the pus and of actinomyces from cultures. The fungus is believed to flourish on corn, hay, and various other grain-bearing plants, and these are the usual means of infection. Infection may take place from animals to man, and there are recorded cases of transmission from one person to another. There is no evidence of transmission of the disease through meat of diseased animals. The disease is commonest in cattle, but it sometimes occurs in horses, hogs, and other animals, as well as in man. In man, on account of occupation, it is commonest in males (seventy-three per cent of cases), and between twenty and thirty years of age. It is seen in a large proportion of cases in those who have to do with the handling of fodder and grain—farmers, coachmen, millers, etc. Infection usually takes place through the gastrointestinal tract, most frequently through a decayed tooth or a lesion in the mouth. It also may occur through the respiratory tract, the skin, or the genital tract.

The lesions of actinomycosis are granulation-tissue tumors which closely resemble in their structure and in their tendency to disintegrate the granulomata of tuberculosis.

The actinomyces or ray fungus is regarded as belonging to the group of streptothrix or cladothrix bacteria. The organism grows in masses which are made up of radiating bulbous threads and spores, and form the characteristic granules of the disease. It grows upon most media and is readily stained.

Diagnosis.—The most characteristic feature of the disease is the appearance of pinhead-sized yellow granules in the pus. Without the finding of these and the discovery of the organism the diagnosis cannot be definitely made. The occurrence of indolent nodular masses which break down with the formation of sinuses, especially about the submaxillary region, should excite suspicion of the disease.

Prognosis.—If the lesions of actinomycosis are so situated that they can readily be treated surgically like other foci of infection, the prognosis is good under appropriate treatment. If left to itself the disease almost always ultimately proves fatal from the involvement of vital organs or from pyæmia.

Treatment.—The local treatment of actinomycosis in the skin is like that of other chronic ulcerative processes. Sinuses should be curetted, the diseased tissues as far as possible removed, and the parts ughly irrigated and dressed frequently with antiseptic solutions. e the removal of the total mass by excision is practicable that may ne. Along with this it is highly probable that the application of s would be beneficial, and Stelwagon refers to favorable results ied in this way.

'hatever the local treatment, it should be combined with the in- l administration of potassium iodide, as first recommended by iassen. This should be given in doses of 40 to 60 grains or more As has been suggested by Ochsner, the best results are gotten potassium iodide in the treatment of actinomycosis by giving it nittently, pushing the drug for a while and then omitting it and ting this process. Its effect is not equally good in all cases, but is no longer doubt of its great value.

evan,[1] from the fact that copper sulphate is powerfully destructive gæ and other low forms of vegetable life, has suggested its use internally and locally in actinomycosis. According to French ists, quoted by Bevan, copper sulphate can be taken in doses of 2 grains a day for six months without any deleterious effect upon the idual. Bevan suggests copper sulphate internally in doses of $\frac{1}{4}$ grain increased to $\frac{1}{2}$ a grain and then to 1 grain, three times a At the same time the affected area should be irrigated with one- nt solution of copper sulphate. The suggestion is ingenious, and iethod should be tried.

III.—MYCETOMA[2]

NONYMS: *Fungus foot of India, Madura foot, Podelcoma, Ulcus grave, Tubercular disease of the foot.*

ycetoma is a disease, endemic in India, produced by a fungus which bably a variety of the ray fungus, and characterized by chronic, ictive ulcerative lesions of the foot or hand.

mptomatology.—Mycetoma begins with the formation of indo- odules in the skin or subcutaneous tissue, similar to those of actin- sis, which break down with the formation of sinuses. The disease ses by the development of new nodules, and as these break down ulcerative, fungoid areas are formed, riddled with sinuses, the lesion causing great swelling and distortion of the part.

Jour. A. M. A., November 11, 1905.

Carter, "On Mycetoma or Fungus Disease of India."—Hatch, Article "Myce- *Encycl. Medica.*—Hewatt, *Lancet*, vol. ii, 1892.—American cases: Hyde, nd Bishop, *Jour. Cut. and Gen.-Urin. Dis.*, 1896 (with bibliography).—Adami irkpatrick, *Trans. Amer. Assn. Phys.*, 1895.—Pope and Lamb, *N. Y. Med.* 1896.—Wright, *Trans. Amer. Assn. Phys.*, 1898, vol. xiii.—Arwine *Jour. Med. Sci.*, vol. cxviii, 1899.

In the discharge from the sinuses there are found in some cases minute black granules (black variety), in others whitish or yellow granules, like fish roe (yellow variety), and rarely reddish granules, like cayenne pepper (red variety). The black granules may, in some cases, be seen under the unbroken skin like gunpowder stains.

The foot is usually attacked; sometimes the leg or hand, and in rare instances the skin of other parts. When it occurs in the foot or leg the disease may spread as far as the ankle, or even to the knee. It said invariably to remain local and never to involve internal organs, b according to Hatch deep glands may be involved and the fungus fou in them.

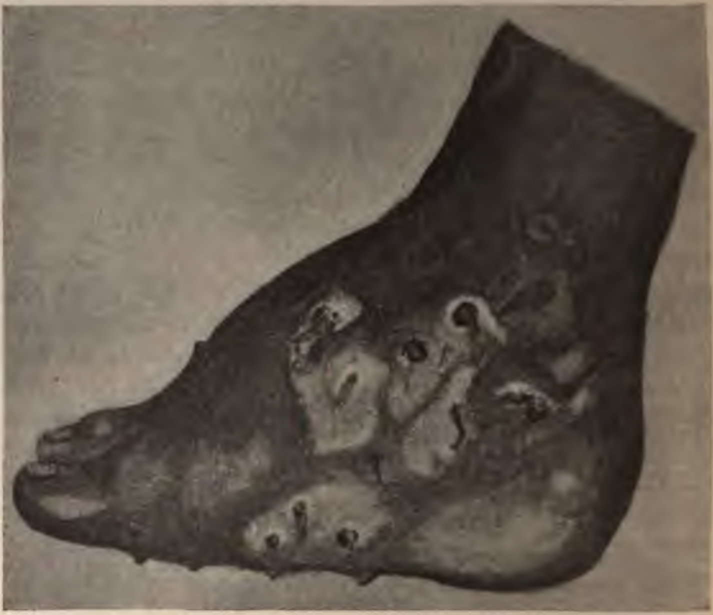

FIG. 195.—MYCETOMA. (L. Raynaud.)

The disease pursues a very slow but progressively destructive cour and ultimately converts the foot into a large, pulpy, suppurating ma of granulation tissue. Complete disintegration of the foot may occu in the course of a year, or the progress may be less rapid; cases may g on for twenty years or more.

The lesions are indolent, and are not spontaneously painful. There is usually no involvement of the general health except when septicæmia develops from secondary infection.

Etiology and Pathology.—The disease is chiefly known from its occurrence in India where it is not uncommon, particularly in Madura.

ive cases have been reported in America, two of these coming from exas. Only one case (Bassini) has been reported in Europe.

The fungus presumably grows upon grain, and the disease occurs ost frequently in those who go barefoot and work in the fields. It supposed to be communicated through injuries.

The disease is produced by a fungus which consists of branching ycelia and spores, and is probably a variety of actinomyces. It is not possible that there is more than one variety of fungus causing the disse. The organism of mycetoma differs from the ray fungus in that stains poorly with acid fuchsin while the ray fungus is highly colored it; it grows very poorly in hydrogen atmosphere, the ray fungus ready; it grows better on vegetable than animal media, the ray fungus ually well on both.

Diagnosis.—The diagnostic features of the disease are its peculiar ographical distribution, its usual location upon the foot, and the enre absence of involvement of the parts commonly affected by actinomysis. Positive diagnosis can be made only upon the discovery of the ack, whitish, or red granules. Its most characteristic clinical feature its peculiar limitation to the foot.

Treatment.—The disease is progressive with no tendency to sponneous healing, and the only successful treatment is thorough removal the diseased tissue. From analogy with actinomycosis potassium dide is indicated internally.

IV.—FAVUS

SYNONYMS: *Honeycomb Ringworm, Tinea favosa, Tinea vera, Tinea lupinosa, Porrigo lupinosa, Porrigo favosa.*

Favus (Latin, *favus*, honeycomb) is an infectious disease of the skin, und typically on the scalp, due to a specific fungus and characterized the occurrence of peculiar saucer-shaped sulphur-yellow crusts.

Symptomatology.—The lesion of favus is a distinctly yellow-coled, saucer-shaped crust—the so-called scutulum (little shield)—which composed of a conglomerate mass of the organism. The crust first pears as a small yellow disk in the epidermis around a hair. It ineases slowly in size, perhaps several months being required for its ll development to the diameter of an eighth to a quarter or half an ch. The crust is thick, slightly concave on the upper surface, and nvex on the under surface. Its usual color is well described as sulphurllow, but from the accumulation of epithelial scales and dirt the older ions may have a dirty or grayish-yellow color. When the lesions are all numerous they have a characteristic musty, mouselike odor. The ngus obtains entrance to the skin in the mouths of the hair follicles and grows in the superficial layers of the epidermis, and to some extent in the hairs. It thus obtains a fixed point of attachment, and as the crust increases in thickness there occur pressure and resultant atrophy of the underlying skin, so that upon removal of the crust there is a marked depression at its site; the crusts lie in these depressions a good deal like stones in soft earth. Along with the pressure atrophy there is a slight inflammatory reaction, probably of mechanical origin, so that the favus crusts are followed by atrophic scarring with destruction of the hair follicles. When the disease is old the characteristic crusts are sometimes absent, and instead there is a diffuse dirty yellowish scaliness of the scalp with characteristic mousy odor.

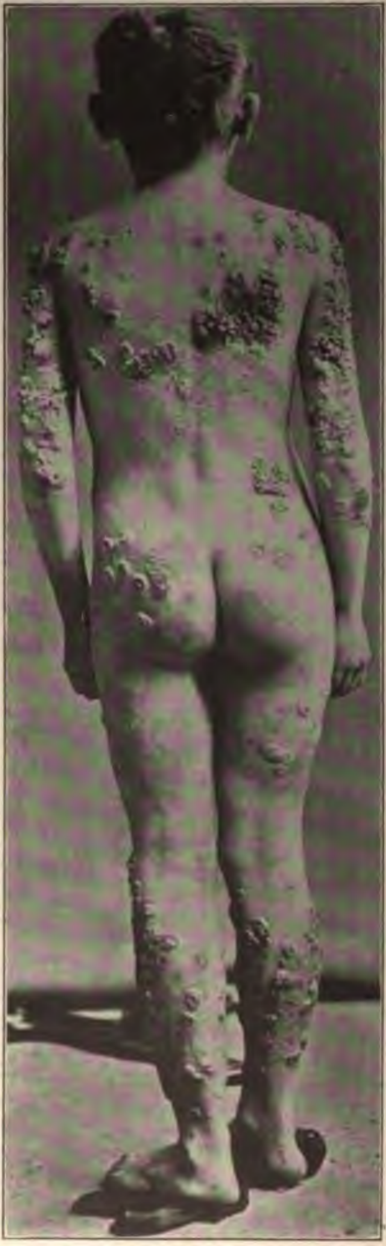
Fig. 196.—Favus of Body. (Grover W. Wende's collection.)

The lesions occur over the scalp with some tendency to irregular grouping. When they coalesce into patches the patches present the characteristics of the individual lesions, and show as a slightly irregular pitted surface which has the honeycomb appearance from which the disease gets its name. The number of lesions varies enormously. The disease as a whole may consist of only a few scutula; ordinarily they are abundant over the entire scalp.

In the early course of the disease the hair over the patches may not be lost, but it is dry, lusterless, and brittle. The scars which result from the favus patches are very characteristic. They are glistening, pink or white, somewhat atrophic and of irregular outline, corresponding with the arrangement of the scutula. They may be quite bald in

, but frequently a hair will grow here and there over the patch, and ɔints, particularly about the borders, there may be clusters of hairs ing. The alopecia of favus is, of course, permanent, and is suf- ıtly distinctive to allow a diagnosis in after years.

avus is usually confined to the scalp, but occasionally it becomes ıdant over the entire body surface, as a rule in connection with the ıse on the scalp. On the body the scutula are of the same character ıose of the scalp, but they may be accompanied by circular ringed hes resembling those of tinea corporis. Crusts develop more rap- upon the body, but as the lanugo hairs offer a less firm site for development, the disease is accompanied by less scarring and is apt to disappear spontaneously. It may rarely involve the nails, hich event the yellow masses may be found deep in the nail sub- es, or the process may be clinically indistinguishable from the homycosis due to tinea trichophytina.

he course of favus is excessively chronic, with no tendency to spon- ous disappearance during childhood. It may disappear of itself ng adolescence or in adult life, but in the frequent cases seen in igrants in America the disease is often present in patients well on lult life, and it may sometimes persist until forty or beyond.

t is practically unaccompanied by subjective symptoms. There is ght amount of itching and feeling of tenderness and stiffness about esions.

Etiology.—Favus may be transmitted from person to person or ı the lower animals, many of which—dogs, cats, rats, mice, rabbits, s, cattle, and horses—are subject to it. It is not infrequently com- icated by cats. The contagion is not very active and it is distinctly sease of those of careless habits and of squalid surroundings. It is ease of childhood, but may develop as late as fifteen.

ts geographical distribution is peculiar. It is common in France in central, southern, and eastern Europe. It is common in Scot- , while in spite of the great poverty of the lowest classes of London, very rare there and in England generally. In the United States it t infrequently seen in immigrants, but is excessively rare in natives.

Bacteriology and Pathology.—The fungus of favus is the achorion ınleinii. This was discovered by Schönlein in 1839, and its patho- c character was established through inoculations by Remak, who it Schönlein's name.

This fungus forms mycelia of narrow ribbonlike branching fila- ts of variable length and thickness, some of them hollow, others sed by transverse septa, often containing spores. l[illegible] numbers of h are intermixed with the mycelia. The s[illegible] rregular in shape and about six micron reproduces by endoconidia, by sprouting, and by formation of unsegmented spindles. Some authorities, as Quincke and Unna, hold that there are several varieties of the fungus—as many as nine having been distinguished; others, as Sabouraud, maintain that there is only one parasite, which varies in appearances on artificial media.

Mewborn [1] has reported a case in which typical favuslike cups occurred on the scrotum and ringworm of the usual genitocrural type occurred upon the inner side of the thigh, from both of which lesions were obtained identical trichophyton cultures. The organism was a megalosporon ectothrix, probably of animal origin.

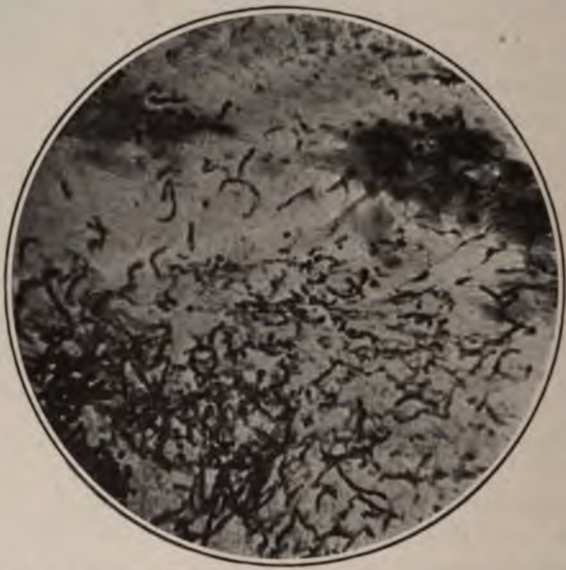

FIG. 197.—SECTION OF FAVUS SCUTULUM, SHOWING SPORES AND MYCELIA OF THE ACHORION SCHÖNLEINII. (Engman.)

The mycelia and spores of favus may be most easily and quickly demonstrated by soaking the hairs and scutula in potassium hydrate solution (ten per cent) after washing with ether; in section they are best stained by polychrome methylene blue, decolorized by 0.1 per cent hydrochloric-acid aniline oil. For staining in the hairs, Adamson's method is perhaps most satisfactory: wash the hairs five minutes in ether, remove the ether with alcohol, and dry on a slide. Stain for ten minutes to an hour with aniline gentian violet, heating slightly; wash and add Gram's iodine solution for five minutes. Pour off the iodine solution, and differentiate under microscope control with aniline oil; clear in xylol and mount in balsam.

The fungus grows readily on glycerine- or glucose-agar and on potato, colonies appearing in about a week. They are powdery, with downy fringe and central elevation which becomes corrugated and wrinkled and of a brownish tinge.

The fungus is to be found in a newly infected hair between the root sheath and the hair follicle; it then penetrates the cuticle and spreads through the hair itself, the mycelia being located chiefly at the center and extending parallel with the hair down to near the root. The mycelial threads are 2 to 3 microns wide, with joints 10 to 15 microns long, and divide dichotomously; spores are not, as a rule, present in the mycelia. The opacity of the hairs is due to replacing of the filaments by air.

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[1] *Jour. Cut. Dis.*, 1903.

In the epidermis the fungus grows chiefly in the corneous layer, but may penetrate to the corium. It forms a mass of interlacing filaments containing many spores, covered by a thin layer of corneal cells. Frequently the scutulum is penetrated by a hair, and the poor nourishment obtainable in the center of the mass results in a cup-shaped central depression and the accumulation in the center of a mass of spores. There are usually proliferation of the prickle-cell layer beneath the scutulum, infiltration of plasma cells, and some degeneration of the fibrous elements in the corium, as a result of which an atrophic condition ensues upon healing. There may be, from the irritation of the fungus or its toxin, an inflammatory hyperæmia and infiltration in the corium with resulting œdema and vesiculation of the epidermis and crust formation.

Diagnosis.—The sulphur-yellow crust of favus is characteristic, and when present renders the diagnosis easy. The irregular, shiny areas of alopecia with scarring are also very suggestive. In cases in which the disease is not very active, and in which no typical crusts are present, the existence of yellowish scales and the peculiar mousy odor give a clew to the diagnosis. In such a case if the patient is told to use no application and not to wash the scalp for ten days, beginning typical crusts will likely be found. In a doubtful case microscopical examination is sufficient to establish the diagnosis.

The irregular atrophic bald patches, independent of the characteristic yellow of the crusts when they are present, are sufficient to distinguish the disease from psoriasis, ringworm, and seborrhœic eczema.

Lupus erythematosus may produce scars on the scalp resembling those of favus, but the scars, while of varying shape, are of more regular outline, and are not the wholly irregular scars of favus. If active, the lesions of lupus erythematosus are distinctly inflamed, itch considerably on the scalp, and show grayish or white scales. Other lesions also are to be found upon the face.

Prognosis.—Favus on nonhairy surfaces is not difficult to cure. On the scalp it is as difficult as ringworm. The prognosis is about the same as that of ringworm on the scalp, but, as the patients are usually of the class that will not submit to the long treatment necessary, it is rarely cured until it cures itself.

Treatment.—In the treatment of favus the scalp should first be freed from crusts by emollient or oily applications. After this the treatment is along exactly the same lines as the treatment of ringworm of the scalp (*q. v.*). The same general precautions against the spread of the disease must be taken, and the same patience and thoroughness in carrying out the treatment are necessary. X-rays, used after the same plan as in ringworm of the scalp, offer the best method of treatment.

V.—TINEA TRICHOPHYTINA[1]

SYNONYMS: *Ringworm, Trichophytosis, Dermatomycosis trichophytina.*

Tinea trichophytina, or ringworm, is a local infectious disease of the skin produced by trichophyton fungi.

In 1843 Grüby discovered trichophyton fungi in ringworm of the scalp, and, as Sabouraud has pointed out, recognized different varieties of fungi. In 1844 Malmsten independently discovered a trichophyton fungus in ringworm of the scalp. Although Grüby's observations were confirmed by Bazin in 1853, the work was entirely overlooked later so far as any differentiation of the fungi of ringworm was concerned, and ringworm was regarded as due to one fungus until the recent brilliant investigations of Sabouraud. Preceding Sabouraud, Furthmann and Neebe, in 1891, suggested that ringworm was produced by different varieties of trichophyton fungi. In 1892 Sabouraud published his now classical researches which definitely established the multiplicity of fungi found in ringworm.

Symptomatology.—The organisms of ringworm grow by preference in horny epithelium, so that the manifestations of the disease are often entirely confined to changes in the hairs and in the epidermis. When the growth is confined to the horny epidermis and hairs, it may be almost unaccompanied by inflammatory reaction; on the other hand, if the organism penetrates into the deeper layers of the skin and connective ssue, as it may, its growth is accompanied by marked inflammatory action.

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[1] Thin, *Brit. Med. Jour.*, 1889.—Furthmann and Neebe, "Vier Trichophytonarten," *Monatshefte*, 1891, vol. xiii.—Duhring, *Amer. Jour. Med. Sci.*, vol. ciii, 1892.—Sabouraud, "Contribution à l'étude de la trichophytie humaine," *Annales*, 1892; "Les trichophyties humaines," Paris, 1894.—Roberts, Leslie, "Study of the Mould Fungi Parasitic on Man," 1893; *Brit. Med. Jour.*, 1894, ii, and *Jour. Pathol. and Bacteriol.*, vol. iii, 1895–96.—Rosenbach, "Ueber die tieferen eiternden Schimmelerkrankungen der Haut und deren Ursache," Wiesbaden, 1894.—Hartzell, *Jour. Cut. and Gen.-Urin. Dis.*, 1895.—Duhring and Hartzell, *Amer. Jour. Med. Sci.*, 1895, vol. cix.—Colcott Fox and Blaxall, *Jour. Pathol. and Bacteriol.*, vol. iii, 1895–96; *Brit. Jour. Derm.*, 1898; *Brit. Med. Jour.*, 1899, ii.—Morris, Malcolm, "Ringworm in the Light of Recent Research," London, 1898.—Pernet, *Lancet*, October 1, 1898.—White, C. J., *Jour. Cut. and Gen.-Urin. Dis.*, 1899, p. 1.—Corlett, *Jour. A. M. A.*, March 18, 1899.—Stelwagon, *ibid.*, November 23, 1901.—Kessler, *ibid.*, October 25, 1902.—Jackson, *Med. Review*, 1902–3.

The clinical manifestations of ringworm vary greatly under different nditions. These variations are in part due to differences in fungi; the aggerated inflammatory type of lesions is frequently the result of a rge spored fungus, usually an ectothrix of animal origin; the persist- it, very slightly inflammatory or noninflammatory type of lesions is ost frequently the result of the small spored fungus. But these varia- ons are also in part due to the circumstances affecting the develop- ient of the organism, particularly the parts of the body involved.

According to location several clinical varieties of ringworm are cognized:

(1) Ringworm of nonhairy surfaces.

(2) Ringworm of the genitocrural region, and occasionally of the cillæ, where the clinical manifestations of the disease are influenced by ie abundance of warmth and moisture.

(3) Ringworm of the scalp.

(4) Ringworm of the beard.

(5) Ringworm of the nails.

Ringworm of the nails resembles other forms of onychomycosis, and most conveniently taken up with diseases of the nails.

The other varieties of ringworm, while they differ very distinctly their clinical manifestations as a whole, all develop in similar ways, id all of the lesions may, therefore, be studied in considering ringworm nonhairy parts. The only essential clinical difference between the fferent forms of ringworm depends upon whether there is involve- ent of the hairs or not.

Tinea Circinata

Synonyms: *Tinea trichophytina corporis, Trichophytosis corporis, Ringworm of Nonhairy Surfaces, Ringworm of the Body, Ringworm of the Glabrous Skin, Herpes tonsurans, Herpes tonsurans circumscriptus.*

Ringworm of the body begins as a flat reddish papule or slightly ele- ted, circumscribed, circular spot. This spreads peripherally and usu- ly clears up at the center, thus forming a ringed lesion. The ring, iich is the active area of disease, is slightly elevated, inflamed. red, and aly, with a sharp outer border and an inner border which fades out ore gradually toward the center. The center is pink or of normal color, d usually shows slight branny desquamation. Such a ring will develop the size of one or occasionally two or three inches in diameter. When e lesions are multiple they may coalesce by spreading and form cir- iate figures, but as the lesions are not usually very numerous or very rge these figures are not, as a rule, complicated. After reaching its full size the ring may remain stationary for a while, but after several days or two or three weeks it tends to disappear spontaneously. It may undergo involution irregularly so that parts of the lesion disappear completely, while crescentic portions persist. In very rare cases two or three or even four concentric rings of this sort have been observed, forming iris figures.

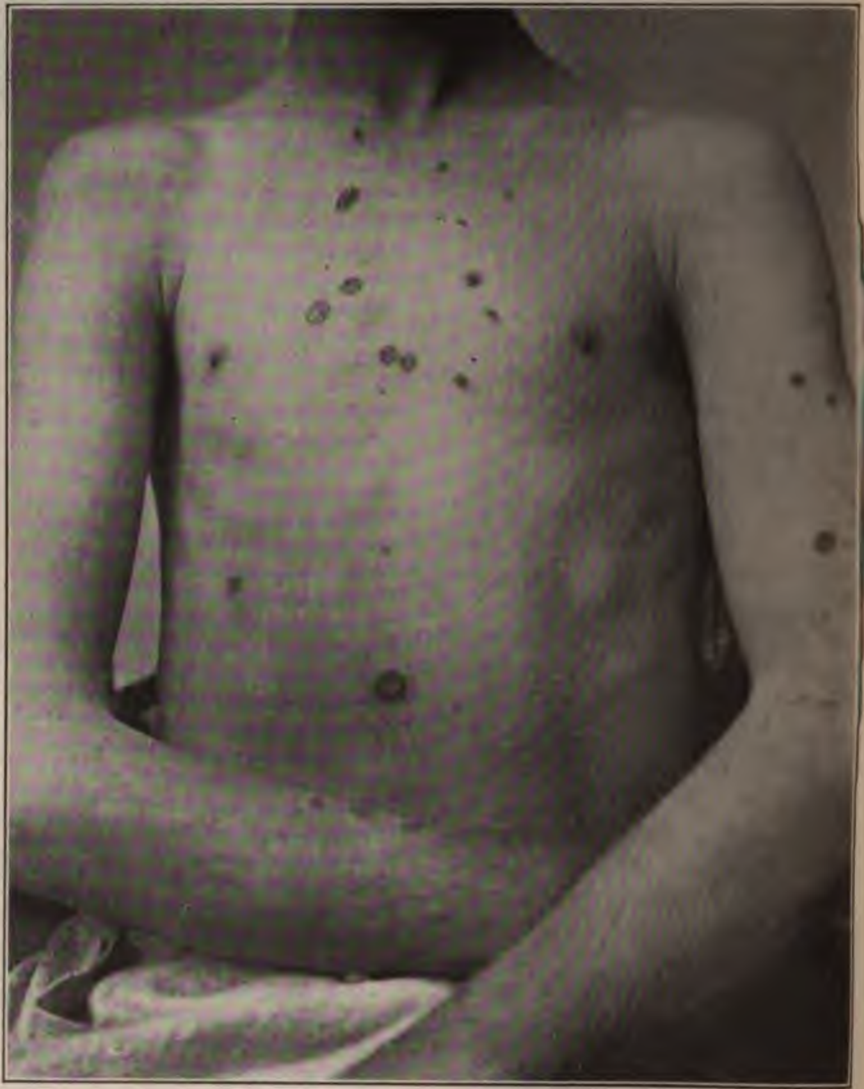

Fig. 198.—Multiple Ringworm of Body, Contracted from a Dog. (Schamberg's collection.)

In other cases lesions begin in the same way as those that produce the rings, but show no tendency to central involution and thus produce, instead of rings, round patches which are slightly scaly, reddish or brownish in color, with rather ill-defined borders, and usually with very little evidence of inflammatory reaction. These lesions may be commingled with typical ring-form lesions.

Occasionally in ring-form lesions the inflammatory process is quite active, so that there is an elevated, red, inflammatory ring, studded with papules and vesicles, or perhaps pustules. The center is of lighter red color, but may be distinctly inflamed and show over its surface papules or pustules. This same intense inflammatory reaction may occur in patches without any tendency to central involution. The patches then present the picture of acute dermatitis; they are red, rather infiltrated, and are covered with papules or vesicles, perhaps are weeping. Their character, however, is still indicated by the sharply defined, slightly more elevated border in which the process is, as usual, most active. This form of lesion differs in no essential particular from ringworm of the crural region, yet to be considered.

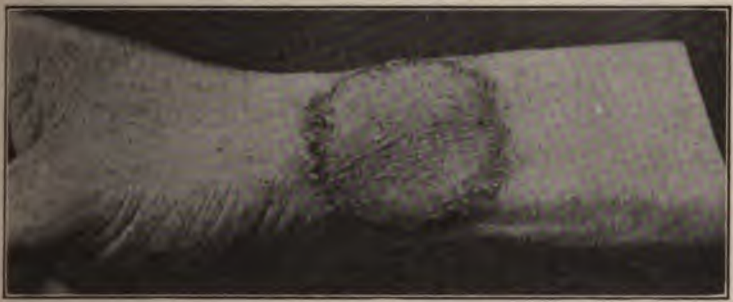

FIG. 199.—TINEA CIRCINATA, PUSTULAR, MEGALOSPORON ECTOTHRIX. (Anthony's collection.)

In rare cases the inflammatory reaction extends much more deeply and produces swollen, elevated, heaped-up masses of granulation tissue similar to the fungoid lesions seen in other forms of vegetating dermatitis. These lesions are sharply limited, from half to two inches or more in diameter, reddish or bright red in color, and may be covered with epidermis or may be denuded and weeping. The hair follicles in them are dilated, and have a greater or less discharge of sero-purulent or purulent fluid. Occasionally in these cases the tendency to central involution may manifest itself, and then there will be formed an elevated border of hypertrophic granulations with a clearing center. At times these conglomerate masses of granulations produce boggy swellings with enormously dilated follicles from which there is an abundant discharge of pus, and thus more or less closely simulate carbuncles. These hypertrophic lesions are the most extreme manifestation of ringworm; they are exactly the same as the lesions on the scalp known as kerion. They have been described as conglomerate *suppurative perifolliculitis*, as *agminate folliculitis*, and as *granuloma trichophyticum*. They are usually produced by the megalosporon ectothrix.

The manifestations of ringworm on the body are usually limited to one, or two, or three lesions; occasionally there may be five, or ten, or even more, but this is not common. Rarely ringworm, instead of being confined to a few well-defined lesions, occurs in numerous small, slightly inflammatory, scaly lesions, generalized over the body and with no tendency to ring formation. These cases may so closely resemble pityriasis rosea that only a microscopical examination will differentiate them. Doubtless it is cases of this sort which led the Vienna school to describe as a form of tinea, under the name herpes tonsurans maculosus et squamosus, the disease which we recognize as pityriasis rosea.

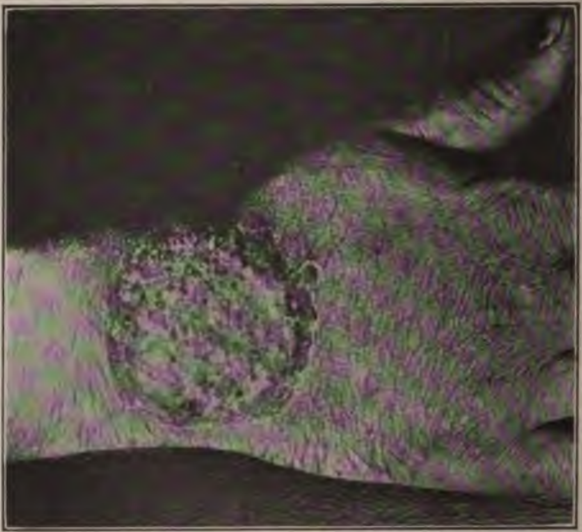
FIG. 200.—AGMINATE FOLLICULITIS. (Heidingsfeld's collection.)

Ringworm may occur anywhere on the body, but its commonest sites are the face, the neck, the hands, and the forearms. When it occurs near the mouth or vulva it may spread as inflammatory lesions to the mucous membranes. Occasionally ringworm appears on the palms; it then manifests itself as ringed or crescentic figures with an actively inflammatory, papulo-squamous or papulo-vesicula border and with less inflamed center. These patch sometimes close simulate scalin papular syphilid of the palms, and may require a careful microscopical examination f a differential diagnosis. Ringworm may also occur upon the sol producing lesions similar to those upon the palms, or developing the result of pressure, into flattened, horny, slightly inflamed callosi

TINEA CRURIS

NONYMS: *Tinea trichophytina cruris, Tinea circinata cruris, Eczema marginatum.*

his name is given to ringworm as it occurs around the genitals. It
s like ordinary patches of ringworm of the body, but from the
able conditions for the growth of the fungi it is accompanied by
inflammatory symptoms. There is produced an acute, slightly

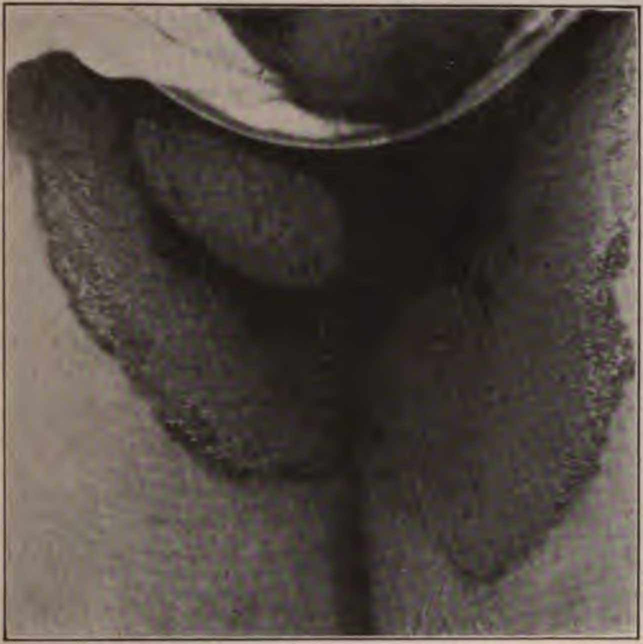

FIG. 201.—TINEA CRURIS. (Grover W. Wende's collection.)

rated papulo-vesicular dermatitis over the involved area. It differs
ordinary dermatitis of these parts by the slightly greater infiltra-
of the patches and the sharp, circinate borders which, as in ring-
lesions elsewhere, are elevated and somewhat more active than the
ior of the patch. The border is usually polycyclic, because the lesion
ade up of confluent circular lesions, and frequently typical ringed
as as they occur on other parts of the body will be found as satel-
This form of ringworm may extend widely upon the thighs, on
bdomen, in the genitocrural fold, and upon the nates. It may also
d upon the mucous membrane of the vulva. The same form of
vorm is seen, but less frequently, in the axillæ.

TINEA TONSURANS

SYNONYMS: *Ringworm of the Scalp, Herpes tonsurans, Tinea tondens, Porrigo furfurans.*

In ringworm of the scalp there are added to the ordinary features of ringworm as it occurs on nonhairy parts the symptoms which result from the destruction of the hairs through their invasion by the fungus. The disease begins as a red papule or a scaly red spot around a hair follicle. This spreads peripherally with the formation of a round, sharply circumscribed, scaly, pinkish or gray patch. At first the hair shows no signs of involvement, but by the time the lesion reaches the size of a small coin the fungus has also penetrated into the follicles and the hair manifests evidence of interference with its nutrition. As the disease persists the hairs become lusterless and brittle and break off, leaving the patch more or less bald. These bald patches are usually slightly infiltrated and covered sparsely by yellowish-gray scales. On inspection with the hand glass the patches are seen to be studded with broken, bent, twisted hair stumps which stick out of the follicles in all directions. The affected area, with its broken, twisted, and disordered hair stumps may be compared to a microscopic forest over which a tornado has passed. The stumps upon traction break off just within the follicles, and the part which is pulled out is usually covered with an opalescent sheath chiefly composed of ringworm spores. The empty follicles are prominent where the hairs have fallen out.

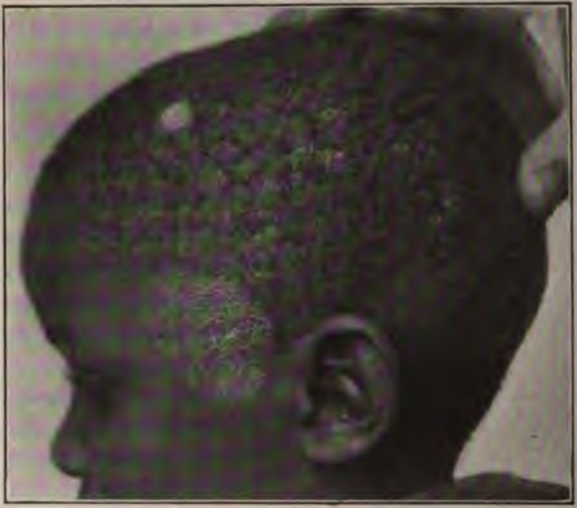

FIG. 202.—RINGWORM OF FACE AND SCALP. (Schamberg's collection.)

As a rule in ringworm the alopecia is not complete. In early patches, and where the process remains superficial, there may be almost no loss of hair, and over the typical bald patches of ringworm there are usually in addition to the hair stumps a greater or less number of dull and brittle hairs. In some cases there is rapid and complete outfall of the hair. The bald spots so produced are sharply circumscribed, and except for slight scaliness may closely simulate alopecia areata. Indeed, in rare cases they become bald and perfectly smooth, so that a diagnosis from alopecia areata can be made only by the discovery of the ringworm fungus in the scales or hairs at the border of the patches. In some cases the spots become almost or completely bald, but the hairs break off at the mouths of the follicles, and thus dot the area with black points—the so-called black-dot ringworm.

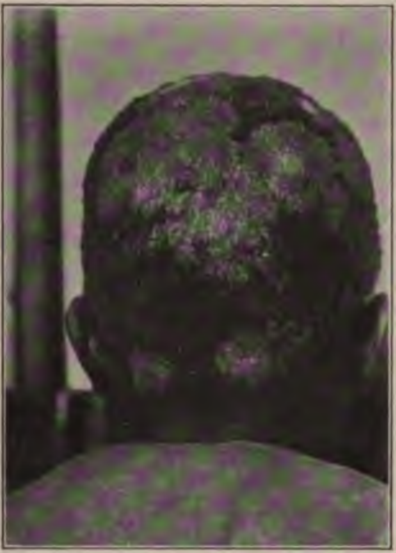
FIG. 203.—TINEA TONSURANS. (Hartzell's collection.)

The single patches on the scalp do not attain a size much above an inch to an inch and a half in diameter, but by the coalescence of adjacent patches large diseased areas are formed. After the disease has existed for months almost the entire scalp may be involved.

Ordinarily the inflammatory reaction in ringworm of the scalp is very slight. It may be absent, but is usually sufficient to cause slight pinkness and infiltration of the patches. Occasionally the inflammatory reaction is more active, and there may be the formation of papules, vesicles, or pustules at the border of the patches. Rarely a single patch of ringworm will develop the hypertrophic area of conglomerate folliculitis which is known as *kerion.* These lesions are exactly like the hypertrophic lesions of ringworm on nonhairy parts, except for the greater prominence of the

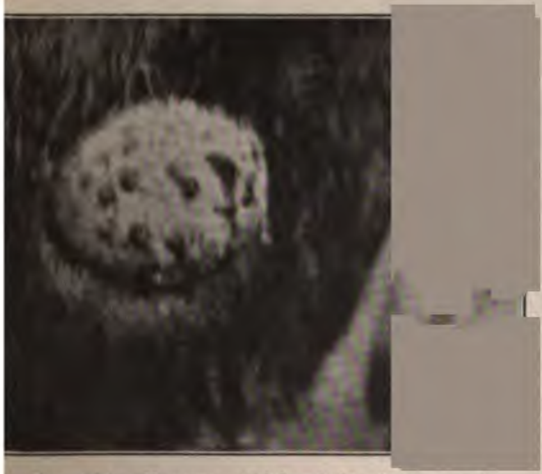
FIG. 204.—KERION. (Lieberthal's photograph.)

hair follicles. At first the hairs stick loosely in the gaping follicles, but they are finally thrown off. The patches of kerion usually result in permanent destruction of the follicles and permanent baldness. The other lesions of ringworm of the scalp do not result in permanent baldness.

TINEA SYCOSIS

SYNONYMS: *Ringworm of the Beard, Parasitic sycosis.*

Ringworm of the beard may either remain superficial or involve the tissues deeply. When it remains superficial it presents in general the characteristics of ringworm of the scalp or of nonhairy surfaces, but is of an inflammatory type. The border is, as usual, sharply defined and elevated, and the most active part of the disease, but the process, while less active at the center, is still quite evident. The hairs are usually less involved than in similar lesions upon the scalp, but they show to greater or less extent the characteristic nutritional changes. Usually they come out easily, showing a sheath of spores around the hairs, or they may fall out spontaneously. In some cases the disease remains quite superficial, spreads peripherally with central clearing, and the hairs remain firm.

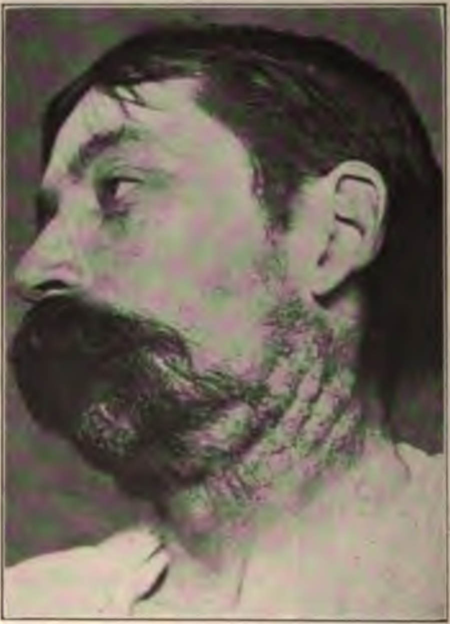
FIG. 205.—TINEA SYCOSIS CONTRACTED FROM A HORSE. (Author's collection.)

Ringworm of the beard may pursue the mild course outlined above with little disposition to deep involvement or to suppuration, but it frequently invades the hair follicles and hairs, and through them the corium, and produces deep-seated lesions such as are found in agminate folliculitis or kerion. The affected area is swollen, red, and angry, and is made up of a conglomerate mass of hypertrophic granulation tissue in which are embedded the distended follicles. The hairs at first protrude, but later they fall out, and the follicles become channels for the discharge of pus or sero-purulent fluid. The follicles may be transformed into well-defined abscesses. This severe form of tinea osis may be limited to a small area; more frequently it consists of merous patches over the bearded part of the face, or it may involve entire bearded area. It is usually most exaggerated under the chin. like simple sycosis it rarely involves the upper lip.

SUBJECTIVE SYMPTOMS OF RINGWORM

The various forms of ringworm with slight inflammatory reaction ve practically no subjective symptoms. There may be slight itching. the lesions accompanied by more acute inflammatory reaction, as in gworm about the genitals, the itching may be considerable—about that of eczema of similar intensity. The hypertrophic vegetating ons, like other forms of vegetating dermatitis, are practically painless.

ETIOLOGY

Tinea circinata and tinea capitis are very common diseases; tinea ris is less frequent but not uncommon; tinea sycosis and kerion are tively rare.

Ringworm exists in man and in many animals, and it is transmitted n all of these sources. It is frequently communicated from patient patient through toilet articles and wearing apparel. It sometimes ginates in barber shops, hairdressing establishments, and the so-called uty parlors. It is most commonly transmitted among children by interchange of hats, and by the common use of combs and brushes other toilet articles. Ringworm is not uncommon among cats, dogs, ses, cows, and other hairy domestic animals, and is frequently transted from them to man.

There is, apparently, a difference in susceptibility to ringworm intion. It is possible that the disease is more readily contracted and re stubborn in persons who are not in vigorous health, and, as Saboud's experiments have shown that ringworm can be more easily inocud in persons with alkaline sweat than in those with acid sweat, this tor may have some influence in the transmission of the disease. The eptibility to ringworm infection is greatly influenced by age. The ase is very contagious in children, especially in the form of ringworm he scalp, and is one of the banes of institutions for children. Ringm of the scalp is practically limited to children, and dies out at or re puberty. Its occurrence is excessively rare after adolescence, algh it has been observed as late as the age of fifty-three. Ringm of the nonhairy parts and of the beard may occur at any age, is very rare after fifty.

Bacteriology and Pathology

Of the hyphomycetes or fungi which cause the lesions of ringworm there are three chief varieties: the trichophyton microsporon or microsporon Audouini, the trichophyton megalosporon endothrix, and the trichophyton megalosporon ectothrix. Numerous subvarieties of each of these have been described, especially by Sabouraud, but the distinctions drawn have no practical application, and are of interest to the botanist rather than to the physician. Sabouraud also makes a distinction between the microsporon and the trichophyta, based on the forms of fructification, which is considered untenable by many other writers.

The methods of demonstration for the ringworm fungi are the same as for that of favus. The fungi will grow on most of the common laboratory media, the medium of choice being maltose agar.

Hairs and scales to be inoculated in culture media should first be well washed in absolute alcohol (up to twelve hours) to destroy possible contaminations. The microsporon shows growth in about a week after inoculation on maltose agar in the form of a circular plaque below the surface, of satiny appearance in transmitted light; as the colony grows old aërial hyphæ develop, the center becomes tufted and opaque, grayish concentric rings form, and the margin presents a fine, downy fringe. Trichophyton endothrix appears in six or seven days as a little star with diverging rays, soon becoming powdery; in a couple of weeks a heap is formed in the center which in a month shows a crateriform depression with cracks running over the surface of the culture. Trichophyton ectothrix varies considerably in cultures; it appears in three days as a little star, with rays more solid and more divergent than t. endothrix, which soon becomes covered with white powdery hyphæ.

Trichophyton Microsporon.—The small spored fungus is responsible for about ninety per cent of the cases of ringworm in this country and in England, sixty per cent in Paris, and very few in Italy. It is almost invariably of human origin, animal infections having been reported in very few cases. It occurs chiefly in early childhood; it usually affects the scalp, and the lesions it produces on the nonhairy skin are, as a rule, of the more transient type. It very rarely affects the beard and practically never the nails. It grows in the stratum corneum, causing a dilatation of the vessels of the papillary layer and hyperplasia

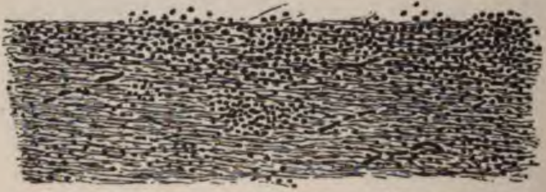

Fig. 206.—Microsporon Audouini (Small-spore Fungus) in Hair. (Engman.)

cells of both epidermis and cutis, which rarely may assume the ter of a granulomatous infiltration with plasma and multinu- l cells. Œdema and faulty cornifications of the epidermis are ed, resulting in and vesicula-

m the epider- out the mouth hair follicle the radually spread ard into the and penetrate ticle above the perhaps by the of a keratolytic proteolytic fer- both of which oduced by the rm fungi. The a spread down- oward the bulb, g a sort of around it, and d within and of the shaft, splitting off the cuticle, and forming the whitish . The mycelia coating the hair break up into segments, which rge, swollen, arranged in rows or groups and known as giant , and these divide into smaller spores, forming a mosaic arrange- around the hair and replacing the cuticle.

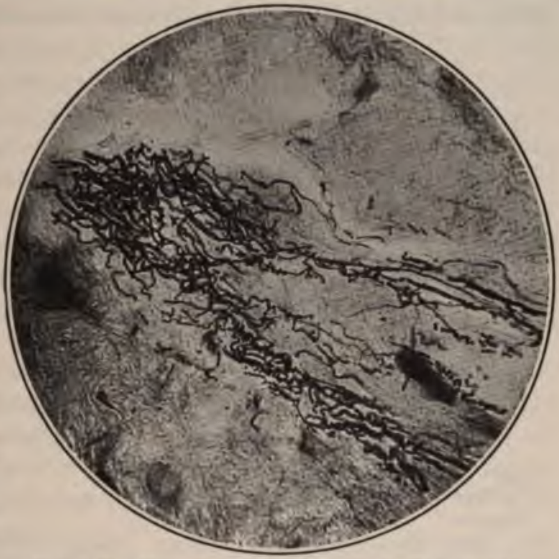

FIG. 207.—MICROSPORON AUDOUINI (SMALL-SPORE) IN TISSUE ABOUT FOLLICLE. (Engman.)

ichophyton endothrix causes only a small percentage of scalp rms in this country and England, though more than trychophyton rix; in Paris it occurs in forty per cent of these cases. It is almost y of human origin, one case being reported in a cat. It affects s. Its lesions on the skin are in general less severe and of shorter on than those of trichophyton ectothrix. It is rarely found in , more frequently in onychomycosis. It may cause kerion.

the skin the pathology of this form of ringworm is entirely simi- that produced by the microsporon. In the hair follicle it is first n the form of spores encircling the hair, which later disappear. reads of mycelia erode the cuticle just above the bulb and enter ir, within which they increase, dividing dichotomously, until aft is filled with interlacing filaments, which break up into celike rows of spores, that are about one and a half to twice as large as those of the microsporon, doubly contoured, and often rectangular.

Trichophyton ectothrix may affect any age; it is rare on the scalp; it is somewhat more frequent on the nonhairy skin than the endothrix, and it is in almost all cases responsible for tinea sycosis and tinea unguium. In many cases where it affects the nails, and in all other localities, it is associated with more or less suppuration, which is in part due to the pyogenic action of the fungus itself, and in part to secondary infection. Trichophyton ectothrix is usually of animal origin, practically all of the domestic animals being subject to it, but it is also communicated from man.

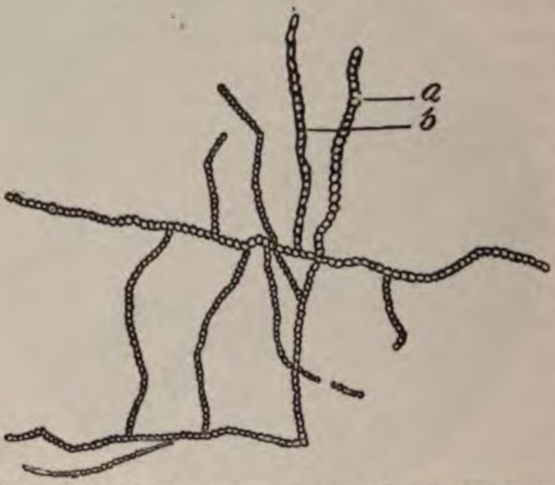

FIG. 208.—MEGALOSPORON ECTOTHRIX (LARGE-SPORE FUNGUS) FROM NAIL SCRAPINGS. *a* and *b*, Filaments of very large spores. (Engman.)

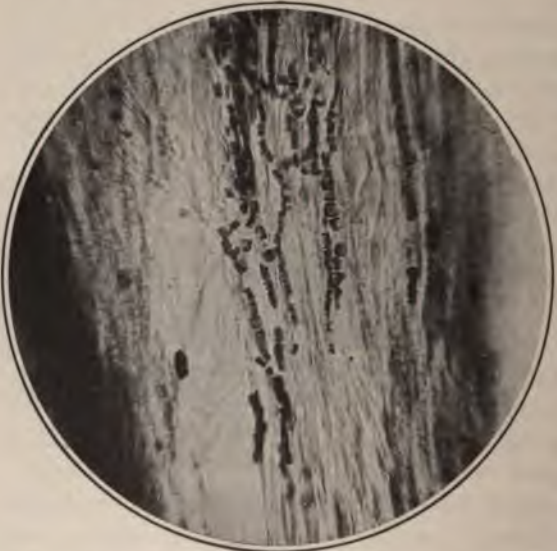

FIG. 209.—MEGALOSPORON ECTOTHRIX (LARGE-SPORE FUNGUS) IN HAIR. (Engman.)

The fungus invades the hair as does the microsporon; the organisms occasionally penetrate the shaft, and chains of spores may be seen within the hair (endo-ectothrix); but they are chiefly situated outside the cuticle, forming a mosaic of spor which appear as sheath fringed at t root. The spores a variable in size (4— microns), often re angular, and lon than they are broa

Both in the scalp and elsewhere this fungus has a tendency to excite omparatively severe inflammatory reaction. In its simpler form this ction constitutes a purulent or sero-purulent folliculitis; where sevl or many neighboring follicles are affected or where the reaction ends into the perifollicular tissues, there frequently results a marked ema of corium and epidermis, with hyperplasia of the cells of both, iltration with plasma cells and round cells, and formation of giant ls, producing a soft, boggy, granulomatous mass, raised above the surnding surface. Variations in the locality affected and in the severity se the process to result in the appearances seen in simple and conmerate folliculitis, in tinea sycosis, and in kerion. In its essence it a hyperplastic reaction in every way similar to that produced by other ogenic microörganisms.

DIAGNOSIS

Ringworm of the nonhairy parts offers little difficulty in diagnosis. e sharply defined, abrupt outer border, the ring form of the lesion h involution at the center, or, if this internal involution has not ocred, the greater activity of the disease at the border, are characterc features. In doubtful cases the diagnosis can easily be confirmed by mination for the fungus.

Seborrhœic eczema, especially as it occurs over the sternum, may be fused with ringworm. In seborrhœic eczema there is usually more ling, and the scales are of a yellowish, greasy type, the infiltration is re equally diffuse throughout the patch and is greater, and there is the narrow border of activity characteristic of ringworm.

Ringworm of the axillæ and of the genitocrural region differs from ema of these parts by its sharply defined elevated active border, ater infiltration, and less active inflammatory symptoms. From seboreic eczema of these parts it differs in its sharply defined borders, the ater and more diffuse infiltration of the surface, less tendency to the mation of scales, and, if scales occur, the absence of the greasy charer of those of seborrhœic eczema.

Ringworm of the scalp may be confused with seborrhœic eczema, psosis, favus, and alopecia areata. Its distinction from favus has been en up under that disease. In seborrhœic eczema and psoriasis of the lp there is much greater scaling, and if there is any effect upon the r it is in the production of gradual alopecia. There is entire absence the broken hairs and stumps characteristic of tinea.

Ordinary forms of tinea capitis differ from alopecia areata in the sence of hair stumps and scales. The very rare cases of perfectly d, smooth tinea require microscopical demonstration of the fungus differentiate them from alopecia areata.

The lesions of kerion and of agminate folliculitis are characteristic, and confusion is possible only with other forms of infected vegetating dermatitis. From other forms of infected vegetating dermatitis it would usually be distinguished by the presence of typical patches of ringworm. In their absence the establishment of the true character of the lesion might be possible only upon demonstration of the fungus. In these lesions of ringworm the fungus may be very deeply situated, and perhaps escape demonstration until sections of tissue are carefully examined for it. The comparative absence of pain and the absence of necrotic abscess cavities should leave no possibility of confusion with carbuncle, although such confusion has occurred.

PROGNOSIS

The prognosis of ringworm varies greatly in different cases. This is chiefly dependent upon the location of the disease, although ringworm due to the small-spored fungus is somewhat more resistant than that due to the large spored. The condition that makes ringworm intractable is involvement of the hairs; in the skin alone its cure offers no difficulty. When, however, the hairs become deeply involved so that the fungus is out of the reach of local applications the treatment becomes excessively difficult, and until treatment with x-rays was introduced was very unsatisfactory. Often the patients with ringworm of the scalp go until the disease disappears spontaneously in early adolescence. Ringworm of the genitocrural region and of the axillæ must be treated with some persistence, but are not difficult to cure. Ringworm of the beard is more intractable, but can be gotten well under vigorous persistent treatment. Kerion is not difficult to get well.

When ringworm of the ordinary type upon hairy surfaces gets well it leaves no trace, either in loss of hair or in scarring; in the kerion type of lesion there is apt to be permanent bald scarring.

In ringworm of the scalp it is often a very important matter to determine positively if the patient is well, and this is a matter of some difficulty. It is never safe to pronounce ringworm of the scalp well except after the most painstaking examination for the scaly spots and after the fungus has been positively demonstrated to be absent. The presence of scaly spots after active ringworm means that the disease is still present. For a satisfactory examination, in order to overlook no patches, it may be necessary to have the hair clipped short. Even after the patients are apparently well an antiparasitic application should be made to the scalp two or three times weekly for several months.

TREATMENT

The treatment of ringworm is altogether local, although in ringworm the scalp in children lacking in vigor the usual measures should be ried out to build them up. In the local treatment of ringworm we end upon the use of parasiticides. When the disease is superficial se agents act directly upon the fungi, but when it is deeply seated h direct effect is impossible and the results are chiefly dependent, not n the direct action of the parasiticides, but upon their irritating perties and the action of the inflammatory process which they excite destroying the organisms.

In treating scaling noninflammatory lesions of ringworm it is de-ble to clean the surfaces first by scrubbing with green soap. In irri-d lesions of ringworm before beginning treatment with the strong lications that are indicated, it is usually desirable, in order to dimin- the pain which is produced by these, to use soothing measures until irritation is subdued. For such purposes we use the same appli-ons that are used in acute eczema, lotions like calamine lotion or c-acid solution, or bland ointments, like zinc ointment with one- wo-per-cent carbolic acid, resorcin, or ammoniated mercury.

Ringworm of nonhairy parts offers no difficulty of treatment. The lication of any of the stronger parasiticides is sufficient. An alto-er satisfactory plan of treatment is to paint the lesions daily for e or four days, or until some dermatitis is produced, with tincture of ne, full strength or diluted with an equal quantity of alcohol. Other lications which may be used are:

Sodium hyposulphite, 1 part; water, 7 parts; carbolic acid, 3 to 5 cent; fresh sulphurous acid; bichloride, 1 to 2 per cent; sulphur ment; ammoniated mercury ointment, or tar ointment.

Any one of these should be rubbed in vigorously for about five utes at each treatment, and the treatment repeated daily or more uently for several days or until dermatitis is produced.

Sodium hyposulphite, 1:8 in water, can be applied as wet dressings he lesion. A plan suggested by Crocker is to soak the lesions thor-hly in the hyposulphite-of-soda solution, and then in a solution of aric acid in water, 15 grains to the ounce. The double decomposition lts in the formation of nascent sulphur and sulphurous acid, and if soaking has been thorough the effect is relatively deep in the skin.

Tinea Cruris.—At the beginning of treatment of genitocrural ring-m it may be necessary to use soothing applications such as have already suggested. As soon as the surface will permit the use of parasitic applications without undue discomfort they should be be-. The same applications that have been suggested for ringworm of the body are used. One of the best applications is the solution of hyposulphite of soda 1:8, which should be applied freely at least twice a day. It may be combined with the application of solution of tartaric acid in the way referred to under treatment of ringworm of the body. Perfectly fresh sulphurous acid is also a good application, but it must be perfectly fresh and it is painful. Other solutions which may be used are three- to six-per-cent resorcin in water, or 1:500 or 1:250 bichloride in water. Occasionally it may be desirable to use a strong application at the border while at the center less irritating applications are used. Strong applications which are useful under such circumstances are tincture of iodine or solution of bichloride 1 to 3 grains to the ounce of tincture of benzoin or myrrh. In the interim between the applications of the antiparasitic solutions it is well to keep the surfaces softened and protected by a bland ointment, such as zinc ointment or cold cream or vaseline, to which may be added a nonirritating quantity of sulphur or carbolic acid or resorcin. Instead of using lotions as indicated above antiparasitic ointments may be applied several times a day. The strength of these has to be varied according to the amount of irritation which they produce. Among the best of these are sulphur or ammoniated mercury 20 to 60 grains, or chrysarobin 10 to 30 grains, to the ounce of ointment. When any of the applications advised above produce an acute reaction, their use has to be omitted and bland applications used until the reaction subsides, and the treatment thus continued until healing takes place. Even after the surface is apparently well an antiparasitic ointment, in a strength which is not irritating, should be used once or twice daily for several weeks to prevent recurrence.

Tinea Tonsurans.—Ringworm of the scalp is one of the most rebellious of skin affections, and its treatment requires infinite pains and persistence. As in all other diseases for which no remedy is satisfactory, the number of agents recommended for the cure of ringworm is almost beyond enumeration, and none of them is efficient unless the treatment is carried out thoroughly and persistently.

Whatever the treatment pursued in ringworm of the scalp there are certain points of general importance which should be attended to. In the first place a child with ringworm of the scalp is a source of danger to other children with whom it comes in contact, and every precaution should be taken to prevent the spread of the disease. This is particularly the case if the child is in school or is in some institution where it is living with other children. Under the latter condition the only safe course to follow is to remove the child unless it can be under such supervision as will preclude the possibility of infection. The child with ringworm, of course, should not be allowed to use any but its own toilet articles. It should always wear some sort of covering on the head to ion from its hat; a paper cap put inside the hat and taken nd burned and replaced by a new one is best if the child is t. If the child is in an institution with other children, a ige should be used which can be removed only by the

treatment of a case the first important matter is to thor-)se all infected patches by clipping the hair short over them. ng should extend an inch beyond the apparent border of the if the patches are numerous the hair should be kept clipped ly as possible over the entire scalp with the exception, per- fringe of hair at the border of the scalp to hide the baldness is worn. As it is sometimes difficult to find small beginning ›re the hair is long, the safest rule is to keep the hair clipped the entire scalp in all cases. Measures should be taken in prevent the scattering of loose infected hairs. The entire 1 be washed every three or four days, and afterwards greased ointment to keep the loose hairs from being scattered about. mething is gained by using an antiseptic soap, such as the bichloride soap or antiseptic green soap, like the following:

,-naphthol	gr. xl;
·ip. sulphur	ʒi;
·n soap	℥i.

(Stelwagon.)

tion to keeping all of the hair short, particular attention has . to the removal of the hairs of the affected patches. It is ilate as completely as possible the hairs of the diseased patches borders as far out as any loose hairs can be found, but this il and very tedious procedure and cannot be carried out in lren. A substitute for it is the persistent and frequent shav- patches. Even better than shaving is the removal of the hair latory, such as the following:

ium sulphide		ʒiij;
: oxide, dered starch,	āā	ʒijss.

(Brayton.)

first made into a paste with water; it is then spread in a on the surface and left until irritation begins. It should moved and the surface washed thoroughly. This is a strong ıpplication and may be of some benefit beyond its action in he hairs.

er remedial agent is used should be applied vigorously and well beyond the border of the patches. Most of the liquid applications should be firmly rubbed in for two or three minutes and then dabbed on for five minutes longer. If an ointment is used it should be rubbed in thoroughly for five minutes. In the use of antiparasitics for ringworm of the scalp they are applied, as a rule, twice daily, until so much irritation is produced that their further use becomes painful. Then they are stopped and a soothing ointment is used until the irritation subsides, and vigorous treatment is then renewed. The course is repeated with constant attention to general measures until the disease finally disappears.

Nothing is to be gained by trying in rotation the numerous agents which are recommended in ringworm. All that it is possible to do can be done with the few well-tried parasiticides. Chief among these are hyposulphite of soda, the mercurials, sulphur, beta-naphthol, iodine, chrysarobin, and croton oil. For use over the entire scalp, the less irritating applications mixed in ointments, as sulphur, ammoniated mercury, and naphthol, are used. For circumscribed patches we may use the more irritating applications, such as tincture of iodine, chrysarobin, croton oil. Croton oil can only be applied cautiously to small patches, and never in children under six years of age. It produces violent reaction, and unless used cautiously may cause bald spots.

In very young children the scalp lesions yield relatively easily. They can be treated by the application of hyposulphite of soda compresses or of solutions of hyposulphite of soda and tartaric acid as referred to under ringworm of the body, or by the use of ointments containing ammoniated mercury or sulphur 20 to 40 grains, or naphthol 15 to 30 grains, to the ounce. Limited patches may be painted once daily with tincture of iodine until a black crust is formed. In older children tincture of iodine may be applied to limited patches two or three times daily until a crust forms; upon its removal the surface is treated with a bland ointment until the irritation subsides. A more irritating and very efficient application which can be used in the same way is composed of equal parts of carbolic acid, tincture of iodine, and chloral hydrate (Cutler's fluid).

The treatment of ringworm by the use of x-rays was first suggested by Freund, of Vienna, but Sabouraud especially has elaborated the method. Sabouraud, by the accurate measurements of the x-rays, reduces the number of applications of x-rays to very few, to perhaps one to an area, but such a method is not safe except in the hands of the most expert. The principle of the treatment of ringworm with x-rays is to produce a slight reaction in the skin which acts therapeutically both by causing outfall of the hair and by destroying the organisms indirectly through the inflammatory process. The exposures should be repeated cautiously, unless the operator is expert, until an erythema is produced and falling out of the hair takes place. In ordinary hands this result requires a good many applications, daily or on alternate days. After the reaction is produced there should be no further treatment over the area until it has completely subsided. One such reaction may be sufficient for cure of the exposed area, but after the reaction has subsided the treatment may be repeated if necessary. When available the treatment of ringworm of the scalp by x-rays is by far the most satisfactory method of treatment that we have.

Kerion.—The treatment of kerion, as of similar lesions upon other parts of the body, offers little difficulty. The intense inflammatory reaction in the lesions itself kills the organisms, and so the lesions tend to disappear spontaneously. The hairs should be removed, the dilated follicles cleaned of secretion, partly by the use of light pressure and if possible by irrigation of the follicles. These measures should be followed by general irrigation of the lesions and ordinary antiseptic dressings. The lesions should never be incised as one is tempted to do, because there is no free pus in the tissues. After the lesions have been cleaned of pus and secretion by daily irrigations and wet antiseptic dressings they should be treated with antiseptic ointments. A good application is

℞	Carbolic acid	gr. x;
	Sulphur	ʒi;
	Lard or vaseline ....ad	℥i.

Another good application is ammoniated mercury ointment.

Occasionally it is a good plan to paint the lesions once or twice with tincture of iodine or lightly with Cutler's fluid, after which a bland ointment or wet dressing is used.

Tinea Sycosis.—The best treatment of ringworm of the beard is by the use of x-rays. This is applied in the same way as in ringworm of the scalp, producing a reaction and allowing it to subside, and repeating the process if necessary. While this treatment is being carried out the parts should be kept clean by the use of bland ointments and antiseptic irrigations. These should be bland in order that they may not themselves produce irritation and thus confuse the operator so that he cannot tell when x-ray irritation occurs. If this treatment cannot be carried out these cases are treated along the same lines as kerion and ringworm of the scalp, but tinea sycosis is much more intractable than kerion. In the first place the lesions and the entire bearded portion of the face should be kept clean by the free use of soap or better [illegible] of antiseptic solutions. The beard should be kept short, and the diseased hairs should be epilated. The bearded part of the face should be well washed with [illegible] soap or tincture of green soap twice daily, and should then be washed in an antiseptic solution (1: 1,000 bichloride). After this it is a good plan to touch suppurating follicles with tincture of iodine or Cutler's fluid, and then apply a bland wet dressing or ointment. If the parts are merely cleansed with bichloride, ammoniated mercury or a similar antiseptic ointment in strength of 20 to 60 grains to the ounce may be applied. This should be spread on cloths, and its strength must be kept below the point of producing irritation. The treatment may be carried out equally well by the use of sulphur applications: Wash the parts for five minutes in solution of sodium hyposulphite (1: 8), and follow this by the application of a sulphur ointment (from 20 to 50 grains to the ounce). These methods of treatment are successful if persistently carried out, but a successful result in severe cases entails treatment for several months.

VI.—TINEA VERSICOLOR

SYNONYMS: *Pityriasis versicolor, Chromophytosis, Dermatomycosis furfuracea.*

Tinea versicolor is an infectious disease of the skin produced by a specific fungus and characterized by the occurrence on the trunk of persistent, brownish, furfuraceous, noninflammatory patches.

Symptomatology.—The lesions of tinea versicolor begin as small chamois-colored or brownish spots. The spots increase peripherally without any tendency to central involution, so that they form diffuse brownish patches from a sixteenth to half an inch or more in diameter, and as these patches coalesce they form large diffuse areas of irregular shape which have all the characteristics of color, slight scaliness, and unctuous feel of the smaller patches. The patches are usually of a dirty grayish, yellowish, or brown color. Occasionally in individuals of delicate skin there may be slight hyperæmia, which gives the patches a pinkish tinge around the edges. At times when the growth of the fungus is very abundant—this happens especially in warm climates—the patches are of dark-brown color and may even be black (pityriasis nigra). The patches are without elevation, soft, and of greasy feel. There is slight furfuraceous scaling and the scales, which consist of a mixture of fungus and epithelium, are readily scraped off.

As a rule the general effect of the eruption on Caucasians is a dirty yellowish or brownish greasy mottling of the skin. In negroes the slight scaliness of the lesions gives them a grayish appearance of somewhat lighter color than the normal skin.

The eruption of tinea versicolor is usually abundant over the affected areas. The spots increase slowly in number, but since after appearing they persist, the disease in the course of several months becomes extensive over its areas of predilection.

The characteristic location is the front of the trunk, where it is most marked over the sternum, and becomes less abundant as it spreads laterally. It also occurs frequently upon the back of the chest and in front of the axillæ. It may extend to the axillæ, and in occasional cases has been found at the bends of the elbows and knees and in the genitocrural region. Sometimes it spreads upon the neck, but very rarely affects the face. It has been observed in a case of Gottheil's on the scalp, palms, and soles.

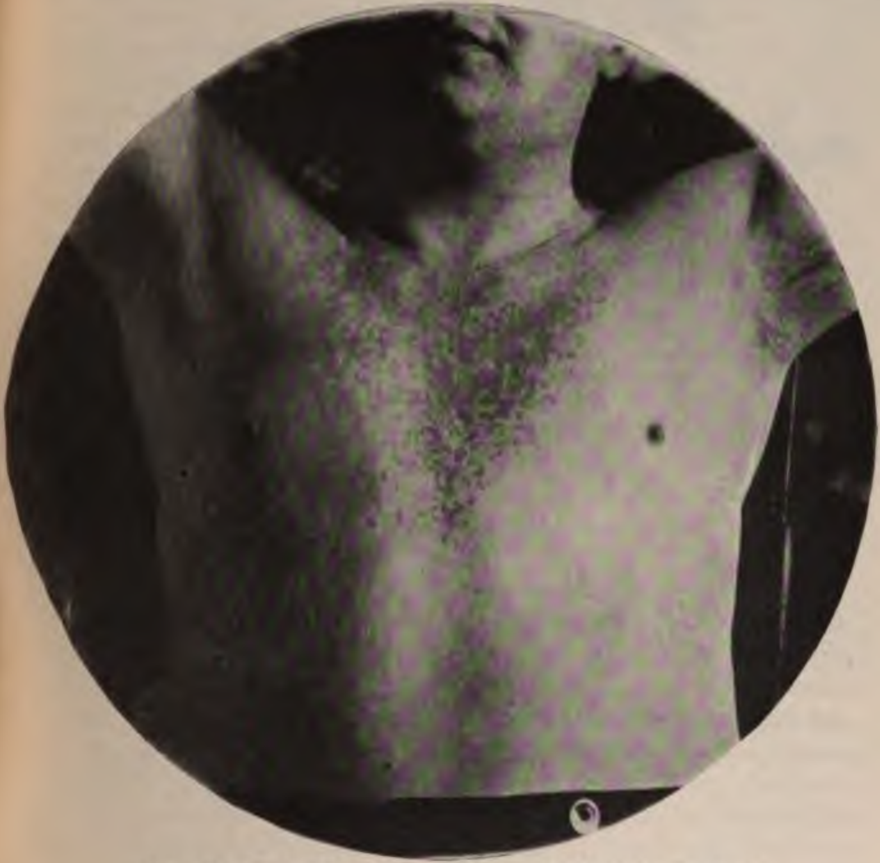

FIG. 210.—TINEA VERSICOLOR. (Grover W. Wende's collection.)

The disease is very persistent, with almost no tendency to spontaneous disappearance and with a persistent tendency to return after apparent cure.

Except when irritated by profuse perspiration and warm weather, when there may be slight itching, it is usually entirely without abnormal sensation.

Etiology.—Tinea versicolor is by no means an uncommon affection, but it is of such trivial character that treatment for it is not ordinarily sought. Hublé found it in sixty-eight per cent of healthy young French soldiers; that, however, is certainly greatly in excess of its frequency in the United States.

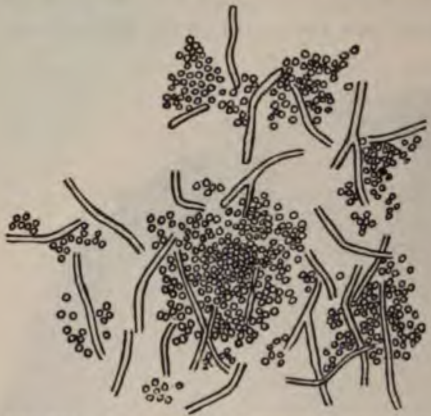

FIG. 211.—MICROSPORON FURFUR, SPORES AND MYCELIA. (Diagrammatic.) (Engman.)

The contagiousness of the disease is very slight, and individual susceptibility is apparently necessary for its production, for it is of common occurrence to see it in one member of a family who has it constantly, while no one else in the household contracts the disease. However, multiple cases in the same family and spread of the disease between married couples are sometimes seen. Inoculation experiments have occasionally been successful, but it is not readily reproduced by experimental inoculations. The individuals who have the disease are usually sparsely built and nearly always perspire freely. It is said to be especially frequent in consumptives.

Bacteriology and Pathology.—Pityriasis versicolor is produced by the microsporon furfur, discovered and named by Eichstedt in 1846. It grows in the superficial part of the horny layer of the epidermis. The spores of the fungus, 2 to 6 microns in diameter, doubly contoured, and with a highly refractile yellowish nucleus, lie in heaps and bunches, around and between which run threads of rather short, usually unbranched mycelia, with sporophores containing conidia at their ends. Matzenauer has cultivated the fungus on Finger's epidermin-agar and reproduced the disease by inoculating the cultures. The fungus is easily demonstrated by soaking scales in caustic-potash solution, and may be stained by any of the aniline dyes.

Diagnosis.—The diagnostic features of the disease are the chamois-yellow color of the spots, the furfuraceous scaling, the peculiar distribution, and the ease with which the scales can be scraped off with a sharp curette or similar instrument. In a doubtful case the mycelia and spores are readily found upon examination.

The diseases with which it may be confused are pityriasis rosea, erythrasma, and seborrhœic dermatitis, but confusion is possible only from ignorance or carelessness.

Prognosis.—It is readily cured by local treatment, but it tends per- tently to recur in susceptible individuals and scrupulous cleanliness no safeguard against it.

Treatment.—Any of the parasiticides recommended for ringworm the body can be used successfully in tinea versicolor. The entirely tisfactory classical treatment consists in the application of sodium posulphite. The skin should be thoroughly scrubbed with soap and ater, using an alkaline soap, such as green soap or one of the better undry soaps, and a flesh brush or a coarse washcloth. This scrubbing done in order to get rid of perspiration and grease and dead epidermis, ut it need not be vigorous enough to cause irritation. After this hypo- lphite of soda solution (1:8) should be applied, or this solution fol- wed by tartaric-acid solution (15 grains to the ounce). These are to applied by wetting a flannel washcloth and rubbing the skin thor- ghly. The treatment should be continued once or twice a day for veral days until the patches disappear. In order to prevent recur- nce the underclothing previously worn must be discarded or thor- ghly sterilized by baking or boiling, and the patient should for sev- al months sponge once a week with sodium-hyposulphite solution or tch closely for the appearance of any patches and attack them gorously.

VII.—ERYTHRASMA

Erythrasma is a disease of the skin due to a specific fungus and char- terized by the appearance of brownish or reddish-brown patches upon rfaces especially exposed to warmth and moisture. It involves par- ularly the axillæ and genitocrural region.

It was described by Burchardt in 1859 and by Bärensprung in 1862.

Symptomatology.—Erythrasma begins in the genitocrural and illary regions with the appearance of reddish-brown or brownish spots. ese are rounded or irregular in shape with well-defined borders, and ve the slight furfuraceous scaliness and the greasy feel which are served in pityriasis versicolor. As the patches enlarge and new patches m they may coalesce into confluent areas which have the character- ics of the smaller patches.

The disease occurs in the genitocrural and axillary regions, and y spread somewhat beyond these. In rare cases it has been found on other flexor surfaces, but it never becomes widely disseminated like ea versicolor. After becoming fully established it does not tend spread further or to disappear spontaneously. It is altogether a ivial affection without other subjective symptoms than perhaps slight hing.

Etiology and Pathology.—Erythrasma is comparatively uncommon in the United States, but is frequent in continental Europe. It is a disease of adult life and is rarely seen before adolescence.

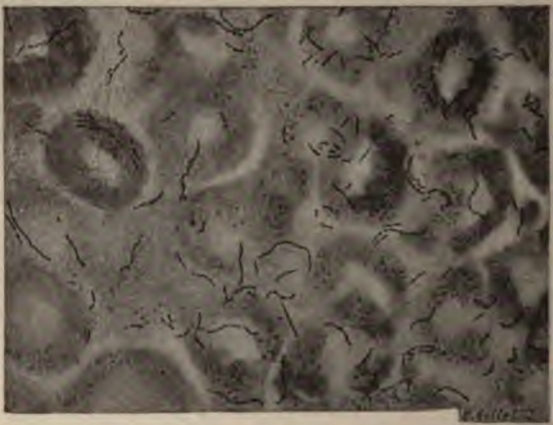

FIG. 212.—MICROSPORON MINUTISSIMUM. Preparation obtained by scraping a patch. (Sabourand.)

Erythrasma is due to the microsporon minutissimum, a very small hyphomycetic fungus; its spores are few in number, and require four to five hundred diameters magnification to be seen. Scales from the disease examined in caustic-potash solution show a dense meshwork of irregularly segmented mycelia, about .7 micron thick, which divides dichotomously. Ducrey and Reale have cultivated the fungus, but attempts at inoculation have been unsuccessful. It grows exclusively in the superficial part of the horny layer of the epidermis.

Diagnosis.—Erythrasma can readily be distinguished from tinea versicolor by the characteristic difference in its sites of development and by its color, which is usually of a reddish tinge and darker than that of tinea versicolor.

In tinea cruris and axillaris the inflammatory features, well developed at least at the border, are sufficient to differentiate it from erythrasma.

Treatment.—The treatment is the same as for tinea versicolor.

VIII.—TINEA IMBRICATA[1]

SYNONYMS: *Tokelau, Bowditch Island, Chinese, India, or Burmese Ringworm, Malabar Itch, Gune, Cascadöe, Le Peta, Herpes desquamans, Scaly Ringworm, circinata tropica.*

Tinea imbricata is a local infectious disease of the skin occurring in tropical countries which is produced by a mycelial fungus and is characterized by peculiar scaly, ringed, concentric lesions.

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[1] Fox, "Narrative of the United States Exploring Expedition, 1838–42, under Command of C. Wilkes," vol. v, p. 401, *American Text-Book.*—Königer, *Virchow's Archiv.*—Anderson, McCall, *Edinburgh Med. Jour.*, 1880, vol. xxvi.—Manson, *Brit. Jour. Derm.*, 1892.—Nieuwenhuis, *Archiv*, vol. xlvi, 1898.—Tribondeau, Abst. in *Brit. Jour. Derm.*, 1899.—Aoki, *Japanische Zeitschrift f. Derm. u. Urol.*, December, 1904.

Symptomatology.—The clinical manifestations of tinea imbricata are due to the very luxuriant growth of a pigmented fungus in and upon the horny epidermis. As described by Manson, about ten days after in-

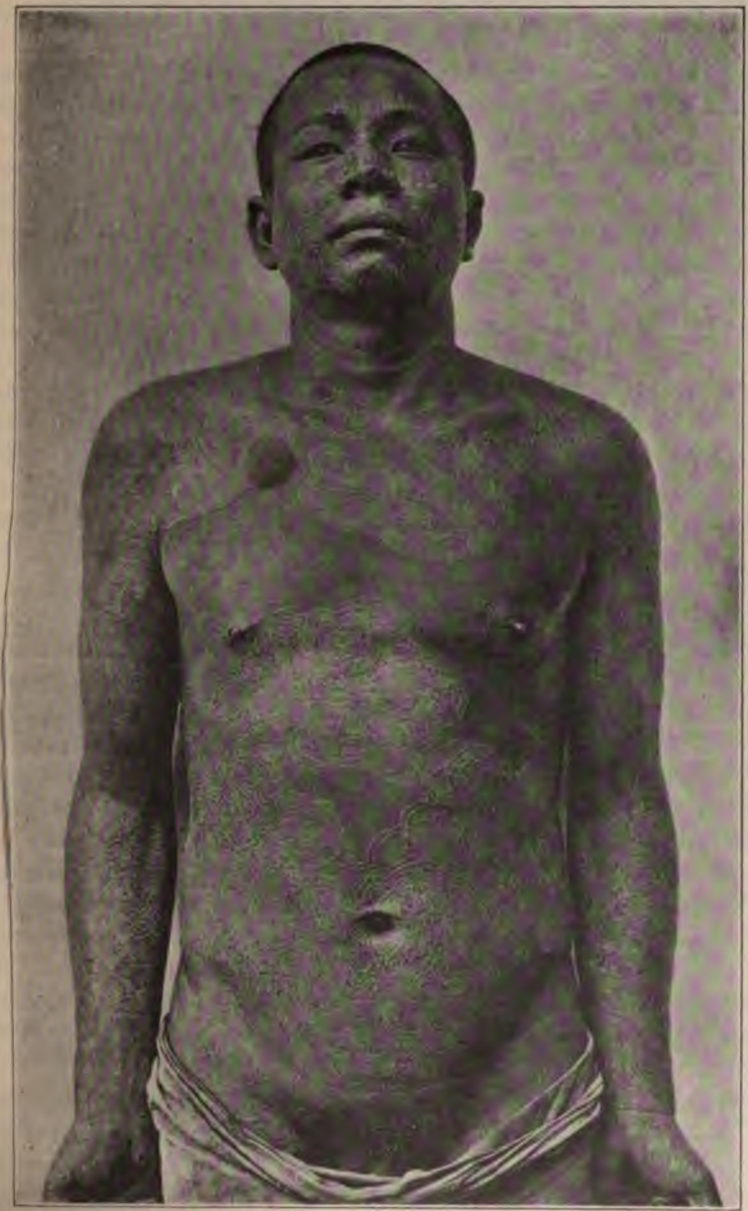

FIG. 213.—TINEA IMBRICATA. (Henggeler.)

oculation of the fungus a minute, slightly elevated brown spot appears, which spreads peripherally, at the end of a week forming a spot about three eighths of an inch in diameter. At this time the epidermis at the center cracks and curls outward, thus forming a lesion whose center is a spot of relatively pale epidermis surrounded by a brownish, slightly scaly ring. Upon its inner border the epidermis of this ring is raised and curled up, while at the outer border it is adherent. There is thus produced a very characteristic and peculiar lesion. The ring gradually spreads peripherally, maintaining a breadth of about one tenth of an inch, and a second brown spot from the growth of the fungus develops at the center and spreads in the same way, forming a second concentric ring about an eighth of an inch within the outer ring, and so the process continues until in thirty to forty days there will be a lesion about an inch and a quarter in diameter and consisting of four or five concentric rings. According to Manson the number of rings may increase beyond this.

The color of the scaly ring is a dark fawn, darker than the lesions of pityriasis versicolor. The color of the lesions is due to the pigment in the fungus and not to true pigmentation of the skin, so that the general effect as regards pigmentation depends upon the amount of pigment in the individual's skin. In very dark individuals the rings will appear of lighter color than the normal skin; in lighter individuals they appear darker.

As the lesions enlarge the scales become more marked and are rather large and thin, a quarter to half an inch in diameter. As the disease becomes older the scales become thicker and larger and of darker color.

The lesions are very numerous, and as they coalesce the involved surface becomes peculiarly figured by coalescing, concentric, iris-form, scaly brown lesions. Manson compares the ringed appearance of an extensive area to watered silk. The disease is persistent, and in the course of time will extend very widely, perhaps involving the entire body surface, but usually leaving the scalp and face free. The growth of the organism remains superficial and it does not commonly involve the hair follicles or the hairs, nor does it involve the nails. Königer, however, describes cases in which the hairs were destroyed.

There is some itching, especially when the patient becomes warm, but there may be no evidences of scratching. In other cases itching may be severe.

Etiology and Pathology.—The disease is found in damp, equable tropical or subtropical climates, and has been observed chiefly in China, the Malay Archipelago, and other parts of the far East. It may develop at any age, but occurs especially in children. It is contagious and is due to a fungus closely resembling the large-spored trichophyton fungus. The fungus grows in the horny epidermis, but according to Manson it grows in much greater abundance than is ever seen in ordinary ringworm. The fungus is readily demonstrated by the same methods used in demonstrating the ringworm fungus.

That the disease is due to a specific fungus different from any of the organisms of ringworm is apparently shown by Manson's inoculation of ringworm trichophyton upon a patient at the same time that he inoculated the fungus of tinea imbricata. The one resulted in a ringworm lesion, the other in a typical lesion of tinea imbricata.

Diagnosis.—The disease can be diagnosticated from ringworm by the fact that the lesions habitually consist of multiple concentric rings, while more than two or three concentric rings have rarely been observed in ringworm. Further, tinea imbricata spreads widely over the surface and the lesions are very numerous, while the lesions of ringworm are few. The dark color of the scales, their large size, and their peculiar loosening on the concave border while they are attached on the convex border are also distinctive characteristics, as is their failure to appear upon the face and scalp.

Treatment.—The disease readily yields to antiparasitic applications, such as are used for ringworm. In order to prevent reinfection after cure the clothing has to be thoroughly disinfected by boiling or completely changed.

IX.—PINTA

Pinta (Spanish, painted) is a contagious disease endemic in tropical America, which is produced by a fungus and is characterized by the occurrence of peculiar persistent discolored patches of the skin.

Symptomatology.—The lesions of pinta are due to the growth of fungi in the skin, and apparently the color of the spots varies according to the site of development of the fungus. When its growth is superficial the spots are grayish, bluish, or black in color; when the growth becomes deeper or when it is deeper from the start, involving the rete mucosum and perhaps the corium, the spots become reddish and the process ultimately destroys the pigment layer so that permanent white atrophic spots are produced. At times in a case the spots are all of one color, while in another case all of the different colors may be present. The spots vary greatly in number, shape, and size. They are slightly or not at all elevated, and at first show slight furfuraceous scaliness which, as the patches become older, increases and becomes lamellar, and the surface becomes rough and dry, not unctuous as in pityriasis versicolor. In the deeper type of lesions, fissuring and ulceration may occur about the flexures. In the superficial type the disease may spread rapidly and become very extensive. If the lesions are of the red or white type their spread is much slower and the disease may remain limited to a few areas.

According to most authorities the hairs of involved areas become thinned, white, and fall out.

The disease usually appears first upon the face and neck, and the areas of predilection are the exposed parts of the body, excepting the palms and soles, which are not involved. In generalized cases the disease may spread to the mucous membranes of the mouth, including the tongue, of the prepuce, and of the vulva. The course of the disease is extremely chronic, and it may persist through life.

The lesions usually itch, and the itching becomes more marked as they grow older and more scaly. The itching is sufficiently intense to cause violent scratching and the changes in the skin which scratching produces.

Etiology and Pathology.—The disease is endemic in tropical America, and probably occurs in other tropical countries. A moist, continuously warm climate is apparently an essential factor in its production. It occurs in both sexes and at any time of life, except early infancy. It is contagious and is favored by careless habits of living and occurs most frequently in the lower classes, but no class is exempt.

Pinta is probably due to a fungus, belonging to the aspergilli or penecillia, described by Gastambide and others as occurring in the corneal layer in the black and blue spots, and in the red and white extending to the rete or even to the corium. The fungus has round or oval spores about 8 microns in diameter, and long fine mycelial filaments which divide dichotomously and form dense reticula connected by a few fine strands. There appear to be a large number (twenty or more) of these pathogenic tropical and semitropical fungi.

Diagnosis.—The clinical picture of the disease is characteristic, and when occurring in endemic localities should offer no difficulty in diagnosis.

Treatment.—The treatment is along the same lines as that for tinea versicolor and other hyphomycetic diseases.

X.—DERMATITIS FROM A MUCOR

Luck has described an eruption of the general appearance and distribution of scabies in which the acarus scabiei could not be found, but in the pustules of which were found mycotic filaments resembling mucor corymbiforme. The affection did not yield to the ordinary applications for scabies, but disappeared under the use of a three-per-cent menthol and salol ointment.

XI.—BATH RINGWORM

Jacobi[1] has described a peculiar form of trichophytosis occurring in insane patients under the treatment with the permanent bath. The lesions, round scaling patches, resemble an abundant eruption of ringworm on the trunk.

XII.—DHOBIE ITCH[2]

Dhobie itch is a term applied in the tropics to a genitocrural dermatitis closely resembling acute tinea cruris. It is not distinctly differentiated from tinea cruris. It is usually accompanied by more intense dermatitis than tinea cruris in temperate climates, but the difference may depend upon climatic conditions. It has been attributed by different observers to various fungi—to the ordinary trichophyton of ringworm, to the microsporon furfur, and to microsporon minutissinum. The disease is infectious, and its name, "Dhobie (Laundryman's) Itch," indicates the impression that it is transmitted in clothing.

XIII.—TINEA INTERSECTA[3]

Under this title Castellani described two cases of what he believes is a peculiar dermatomycosis. The condition began with small, roundish, slightly elevated itching spots on arms, chest, and back. The patches became dark brown, presented a smooth, tense surface, increased in size slowly, and coalesced. Later the surface of the patches became shrivelled superficially and cracks appeared, forming white lines in the brown surface. Later the cracks deepened, the epidermis split, and flaky, loose scales curled up. The fungus showed the characteristics of trycophyton except for the absence of free spores in the preparations examined. The abundant mycelium was composed of long, straight, articulated threads, which were sometimes dichotomous. Endospores and endoconidia were present. He did not succeeed in cultivating the fungus. In contrast to tinea imbricata the eruption did not develop in concentric circles; patches disappeared spontaneously, and the disease was cured without difficulty by the usual antiseptic applications.

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[1] *Archiv*, 1907, B. 84, p. 289.

[2] Manson, "Tropical Diseases," Fourth Edition. Castellani, *Brit. Med. Jour.*, 1905, vol. ii, p. 1277; *Jour. Trop. Med.*, 1905, p. 253.

[3] Castellani, *Jour. Cutan. Dis.*, 1908, p. 405.

XIV.—TINEA ALBIGENEA[1]

Under this title Nieuwenhuis has described a peculiar ringworm which he has studied in the Dutch East Indies. It occurs in large, roundish, white, scaly patches on the palms and soles, and spreads thence to the adjacent skin of the hands and wrists and feet and ankles.

XV.—MYRINGOMYCOSIS[2]

Synonyms: *Otomycosis, Myringomycosis aspergillina, Otitis externa parasitica, Fungous disease of the external ear.*

This is an inflammatory process involving the auditory meatus, the external auditory canal and the drum, which is believed to be due to a specific fungus. It occurs as a superficial inflammatory process which produces a scaly, moist, pultaceous crust of dirty green or brownish color in which there may be yellow, green, or black points. Under this crust the surface is red, moist, or weeping. Occasionally only the meatus is involved, but as a rule the external canal is also involved, and it is thought that the disease usually primarily attacks the drum. The hearing may be impaired by damage to the drum, which may amount even to perforation. The condition is accompanied by itching or stinging, sometimes pain, and a variable degree of deafness. It does not spontaneously disappear. It is to be differentiated from seborrheic dermatitis, which produces a grayish-yellow, greasy scaliness. The condition is believed to be due to infection with aspergillus niger and aspergillus glaucus. It yields to alkaline and weak antiseptic applications. Burnett recommends a one-per-cent solution of sodium hyposulphite, and Lowenberg recommends washing with alcohol, dilute or full strength. The presence of ointments is said to favor the growth of the fungus.

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[1] Nieuwenhuis, *Archiv*, 1908, B. 89, p. 1. (With illustrations and mycology.)

[2] Barclay, "Burnett's System of Diseases of the Ear, Nose, and Throat," vol. i, p. 190.

SECTION VIII

DERMATOSES DUE TO ANIMAL PARASITES[1]

THERE are many animal parasites which may occasionally prey upon the skin. The number of those, however, which are habitual parasites of the skin is relatively few in the temperate zone. Among these, far and away the most important, so far as their rôle in the production of skin diseases is concerned, are the pediculi and the acarus of scabies. Other common parasites of the skin which are the source of much annoyance, but are of relatively slight importance so far as their skin lesions are concerned, are fleas, mosquitoes, bedbugs, and like pests.

According to their mode of attack upon the skin, animal parasites may be divided into two classes:

(1) Those which live outside the skin and attack it from without by biting or stinging.

(2) Those which live in the substance of the skin.

Familiar examples of the first class are pediculi, fleas, mosquitoes, bees, wasps, spiders. Some of these invade the skin for the purpose of obtaining food; others only occasionally attack the skin, and then as a means of defense. In the lesions the reaction which results is not the result of the slight mechanical traumatism produced by the bite or sting, but of an irritating secretion, chiefly formic acid, introduced by the parasite through its wound. The lesions which result from these external parasites all resemble each other, varying chiefly in degree. At the site of the bite or sting there is formed a minute red punctum, into which in some cases the blood wells up and dries into a minute reddish black speck. This punctum at the site of the parasite's wound is the most characteristic feature distinguishing these lesions from lesions of the same kind due to other causes, but it disappears in the course of several hours or a day unless there is a blood crust in it, and so may escape observation.

The lesions produced by stings and bites are inflammatory, œdematous wheals. Their size varies from a papule to a large œdematous swelling, but they are all of the same character, differing only in degree.

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[1] Geber, "Handbook of Skin Diseases," Ziemssen's "System."—Ritchie, "Parasites," *Encyclopedia Medica.*

In a relatively insusceptible individual a flea bite may produce only the punctum, or at most a small inflammatory papule; in a person of average susceptibility, the bite of a flea or mosquito produces a reddish or pale waxy wheal from the size of a pea to that of a finger nail, indistinguishable in appearance, except for the punctum, from the wheal of urticaria, while the sting of a wasp or bee produces a large, swollen, œdematous inflammatory lesion, sometimes pale like the ordinary urticarial wheal, but usually larger, red, swollen, and tense. All of these lesions are, as a rule, evanescent, and disappear in a few hours, leaving usually no trace or a hæmorrhagic punctum. Frequently, however, the itching which results is so intolerable that violent scratching is induced, producing excoriations, papules, and other evidences of traumatic dermatitis, and these manifestations of the scratched skin are the chief objective symptoms of the parasitic diseases of this class.

The lesions resulting from parasites which exist in the skin are quite diverse, and have not enough features in common to allow of any general description.

The duration of all of the dermatoses produced by animal parasites depends upon the duration of the parasites. They do not, like many of the pathogenic bacteria, cause the production of any antibodies that render them self-limiting.

I. DERMATOSES FROM ANIMAL PARASITES WHICH ATTACK THE SKIN FROM WITHOUT

I.—PEDICULOSIS

SYNONYMS.—*Phthiriasis, Morbus pedicularis, Morbus pediculosis, Malis pediculi, Lousiness.*

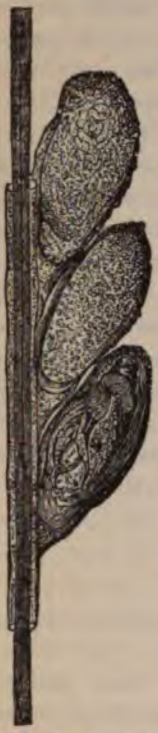
Pediculosis is the term applied to the conditions in which the body becomes infested with pediculi or lice. There are three forms of pediculosis due to three species of pediculi which attack the skin:

(1) Pediculus vestimentorum or pediculus corporis, which lives in the clothing.

(2) Pediculus capitis, which lives in the hair of the scalp.

(3) Pediculus pubis, which lives in the pubic and occasionally in the other short stiff hairs.

All three seek the skin for nourishment, and thus produce their ravages. Each one of these parasites confines itself, as a rule, with remarkable regularity to its particular territory, and is only accidentally and temporarily found elsewhere. In addition to this choice of location, there is also considerable choice exercised by pediculi in their tion of individuals. Certain persons are much more attractive ts than others, and are much more likely to be attacked. Aside n this personal susceptibility of certain individuals, there is an uired susceptibility, especially to pediculosis poris, which comes from cachexia or debility.



. 214.—Ova of Pediculi Capitis. (Kaposi.)

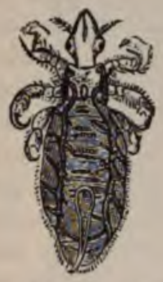


Fig. 215.—Pediculus Capitis. (Küchenmeister.)

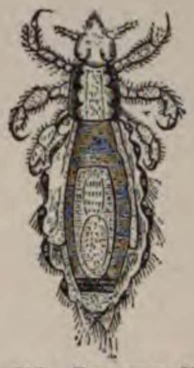


Fig. 216.—Pediculus Vestimentorum. (Küchenmeister.)

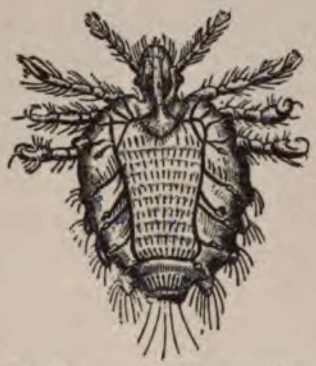


Fig. 217.—Pediculus Pubis. (Schmarda.)

The body louse is in structure like the head louse, but is larger. The l louse is about 1⅕ to 3⅕ mm. long, the body louse from 1⅕ to 4⅕ mm. . They are large enough to be easily seen by the naked eye, but account of their translucent grayish color readily escape detection. h are grayish and have a blackish margin; after feeding they bee, from the blood which they imbibe, of a reddish color.

The crab louse, so called from its form, is broad and short, and es in length from 1 to 2.1 mm., and in addition to the six jointed s and the antennæ common to all these varieties it has eight presile feet, four on either side the abdomen. It is of translucent yish or yellowish color.

All reproduce very rapidly. In the case of the head and pubic lice, the ova or nits are attached to the hair shafts at an acute angle. They are minute yellowish or grayish-white pearllike bodies of regular oval shape, visible to the naked eye and firmly attached to the hairs. They hatch in about a week, and the lice develop within two weeks.

The pediculus feeds by inserting its labium or sucking apparatus into a sweat pore, and in the process of feeding injects an irritating secretion into the bite. When the animal stops feeding, particularly in the case of the pediculus vestimentorum, a minute droplet of blood wells up into the wound and dries into a pinpoint speck. These can be seen upon close examination in most cases of pediculosis corporis, and are the most characteristic feature of the lesions produced by the pediculi. They are not so readily found in pediculosis capitis or pubis.

At the site of each bite of a pediculus there immediately develops a transitory wheal, which itches intensely and which is likely to be excoriated by scratching. In addition, therefore, to the lesions due primarily to the bites, there results dermatitis from scratching, usually a discrete papular dermatitis which is apt to be associated with excoriations and, as a result of secondary infection, pustules and other infected lesions. The objective symptoms are for the most part confined to the locality invaded by the parasite, but may spread beyond it. Itching of reflex origin is usually excited at points of the body not invaded by the parasite, and is frequently general.

PEDICULOSIS CORPORIS

SYNONYMS: *Pediculosis vestimenti seu vestimentorum, Phthiriasis corporis, Vagabond's Disease.*

Symptomatology.—The pediculi vestimentorum live in the clothing, especially about the seams; sometimes, in exaggerated cases, they live on the body and deposit their ova upon the lanugo hair. From the clothing they go down to the skin to forage. As a result, we find the first and the greatest evidences of the ravages of this pediculus at points of contact of the clothing, because these points are most easily reached. The characteristic areas of predilection for the eruption are the shoulders and the upper third of the back, the waist, and the extensor surfaces. In mild beginning cases the lesions may be found in these locations. In severe cases the lesions become generally distributed over all the covered parts of the body, and may amount almost to a confluent dermatitis. The hands and face, however, nearly always remain free.

The eruption of pediculosis corporis is usually one of discrete inflammatory papules with scratched, blood-crusted tips. Nearly always, in addition to the scratched papules, linear scratch marks can be found, with pustules or other suppurating foci as the result of secondary infec-

n. Except for the hæmorrhagic speck in the bites, not to be con- ed with the blood crusts ich cap the scratched pap- s, the lesions are such as y be produced from vio- t scratching from any use, but rarely does any ier dermatosis itch so in- nsely or show such extreme coriations.

The intensity of the anifestations of the der- atitis varies very greatly ith the duration of the af- ction. When the affection recent the eruption con- its of numerous discrete, ratched, inflammatory pap- es, with perhaps some heals and linear excoria- ons. As the condition per- ts the lesions become more undant, secondary infection oduces pustular lesions, d the eruption becomes a dely distributed, abundant, haps confluent dermatitis. extreme cases the skin omes greatly thickened, ltrated, and leathery.

When the disease persists any length of time, espe- lly in the aged, there is ically a good deal of pig- ntation of the skin. This mentation usually consists light brownish spots, but the disease persists month er month the pigmenta- n becomes diffused over trunk and other affected as. In cases of long dura- n the skin may be col-

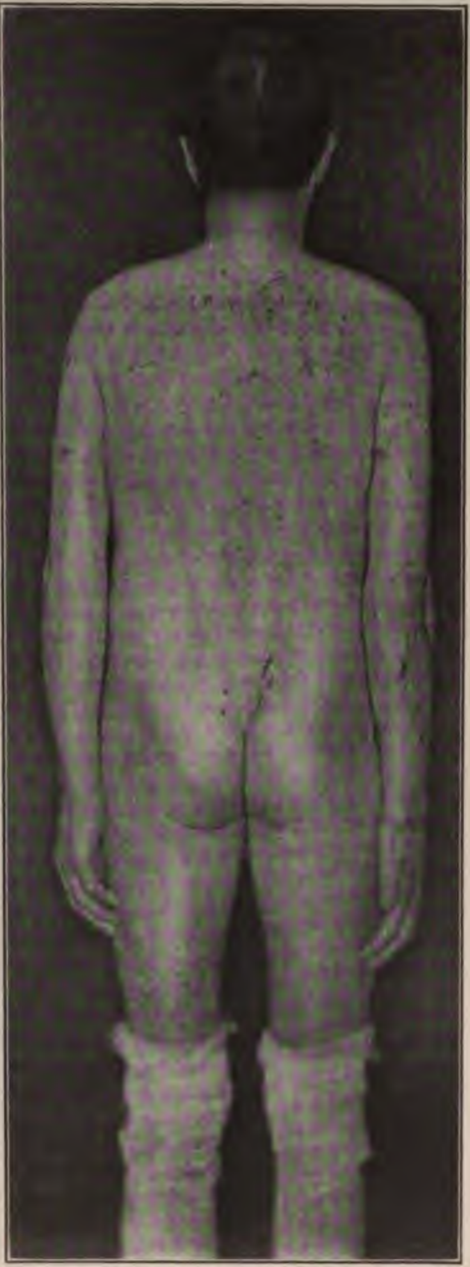


FIG. 218.—PEDICULOSIS CORPORIS.
(Author's collection.)

ored a dark brown, and in extreme cases even black. There seems every reason to believe that this pigmentation is of the same character as that following other chronic inflammatory processes in the skin, but occasionally it has curious features. It may at times be out of proportion to the dermatitis, and Thibierge has found it in the oral mucous membrane. The explanation of this pigmentation of the mucous membrane is not clear; Thibierge suggests that it is due to the deposit of pigment which has been transferred from the lesions in the skin; Bouc that it is due to a toxæmia from the secretion which is injected by parasites.

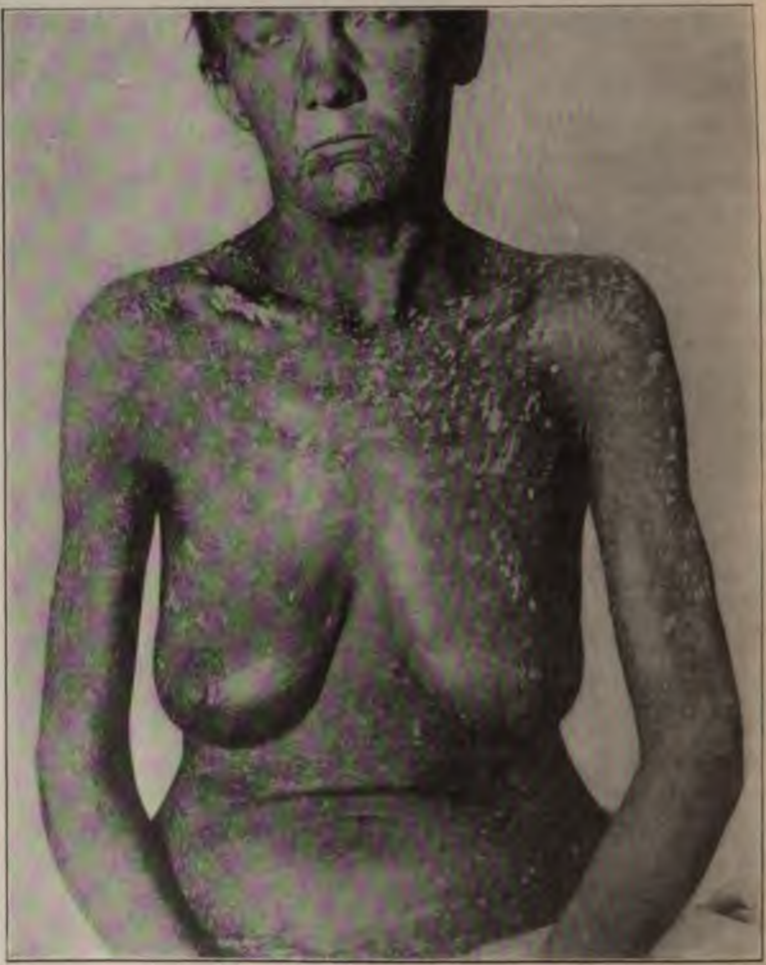


FIG. 219.—PIGMENTATION FROM PEDICULOSIS. (Photographic reproduction from Piffard's Atlas.)

he chief subjective symptom is violent itching, which is not con-
to the sites of the dermatitis but may occur reflexly upon any
of the body. Along with the itching there may be burning and
ication.

n severe cases, especially in children, there may be several degrees
ver, which Payne ascribes to the poison injected by the parasite.
more probably of inflammatory or septic origin.

tiology.—Anyone who is exposed to vermin may contract pedi-
is, but the pediculi do not flourish on vigorous young clean sub-
, and if they become infested the affection either disappears of
or promptly succumbs to cleanliness. Pediculosis is of all dis-
most characteristically the disease of the vagrant and neglected
s. It is rare in children and is uncommon in middle life, but
s typically in the old. In children it is only seen in the most
cted or in those who are in continuous intimate contact with older
ns.

isease, alcoholism, overcrowding, dirt, and neglect are the predis-
g factors. It is contracted by intimate contact, and therefore is
common in winter, and is seen especially in the lodging-house
In addition to the increased susceptibility which comes with
ed health, it is a common observation that among healthy indi-
ls there is a difference in susceptibility. Crocker recounts the
ce of four young medical men, who placed a pediculus in the
le of a table, around which they stood, and found that the louse
s went to the same man regardless of any change in their positions.

iagnosis.—The diagnostic features of pediculosis are the hæmor-
c specks in the bites, the predilection of the eruption for the upper
of the back and shoulders, the age and the class of the patients.
lently scratched skin over the shoulders, with more or less evidences
ratching upon other parts of the body, in an old patient of the
r class is almost pathognomonic of pediculosis. When the features
ed to above are found the diagnosis is confirmed by the finding
morrhagic specks in the skin and pediculi and nits in the clothing.
pediculi are to be searched for in the seams, especially about the

reatment.—The treatment consists in measures to get rid of the
uli. They can be killed in underclothing by boiling, but repeated
g is usually necessary. They are best destroyed by heating the
ng in an oven; the temperature must be carried at least above
oiling point, but need not be high enough to burn the clothing.
the pediculi are destroyed the dermatitis rapidly subsides of itself,
ts disappearance is hastened by the use of soothing applications,
inc-oxide ointment, to which, because of their parasiticidal proper-

ties, it is well to add sulphur and balsam of Peru, equal parts, 15 to 30 grains to the ounce.

To get rid of any pediculi which have otherwise escaped destruction it is well to dust the clothing lightly with washed sulphur, as suggested by Sherwell in the treatment of scabies.

Pediculosis Capitis

Synonyms: *Phthiariasis capitis, Pediculosis capillitii, Head-lousiness.*

Symptomatology.—The pediculus capitis in feeding upon the scalp produces itching, and in very mild cases this may be the only symptom beyond the presence of lice and nits. More often there is some evidence of dermatitis as a result of scratching and secondary infection. This may consist of a few discrete inflammatory papules and patches or suppurating areas, or there may be a diffuse dermatitis with a greater or less amount of suppuration over the entire scalp.

From the exudation of serum and pus the hair is more or less matted together, and in severe cases may be a festering mass. The cases which are seen especially in eastern Europe, and described as *plica polonica*, are the most exaggerated illustrations of the condition. In plica polonica the long hair of women becomes matted together with lice, nits, and dried secretion into a semisolid, tail-like mass. Such cases occur only after long persistence of the disease in the filthiest people, and are practically never seen in America.

The effects of pediculi are usually most intense over the occiput and behind the ears. In severe cases there are apt to be inflammatory lesions from secondary infection on the face and neck. As result of secondary infection, not uncommonly the occipital glands are enlarged and tender, and may suppurate.

The number of pediculi and ova vary greatly in different cases. They may be so few that only the most careful search behind the ears will find the nits. As a rule, both lice and nits are abundant. The itching which accompanies this condition is severe, and is the primary cause of all the objective symptoms.

Etiology.—Pediculosis capitis is a common affection. In its severe forms it is, of course, rarely seen in any but the most careless individuals, but there are probably few children who attend public schools or who mingle freely with other children that have not had pediculosis capitis. The transference of pediculi capitis requires intimate contact, and is usually the result of the closest personal association, the interchange of hats, or the common use of brushes and combs. The disease is seen most frequently in children, and, on account of their long hair, of more frequent occurrence and commonly of severer type in women an in men.

Diagnosis.—The occurrence of pustular dermatitis over the occiput behind the ears, especially in women and children, should always ggest pediculosis. The suspicion must be confirmed by finding the diculi or the ova. The pediculi may not be easy to find; the ova n usually be found behind the ears. They are very easily confused ith minute scales of dandruff; but dandruff scales lie loose in the air, are readily moved along the hairs to which they may be attached, nd are irregular and lamellar and without the regular pear shape f nits.

Treatment.—In pediculosis, if the dermatitis is not severe, vigorous arasiticides may be immediately applied. If the scalp, however, is a ontinuous area of acute dermatitis, it is best first to thoroughly clean p the scalp with soap and water and apply mild parasiticidal ointments for two or three days until the scalp is cleansed of pus and dirt and relieved somewhat of irritation. For this purpose a salve of ammoniated mercury, sulphur, or beta-naphthol, from 15 to 30 grains to the ounce, can be used, and after this the strong parasiticidal application. The applications commonly used are kerosene, corrosive sublimate, and tincture of cocculus indicus. The time-honored plan of treatment is to soak the hair thoroughly in kerosene, or equal parts kerosene and olive oil, liquid vaseline, or linseed oil. Any of the kerosene which runs down upon the face or neck is wiped off to avoid irritation, the scalp enveloped in a loose head bandage, and the patient cautioned to stay away from a lighted lamp or fire. This latter is a necessary precaution to avoid accidents. After twelve to twenty-four hours the kerosene dressing is removed and the scalp thoroughly washed in soap and water. One application is usually sufficient to kill both the lice and nits, but to be safe it is well to make a second application. A much more agreeable method is to thoroughly wash the hair and scalp first with soap and water, and then with a solution of bichloride and water, 1:500 or 1:250. After this preliminary washing the hair and scalp should be wet thoroughly with the solution once or twice a day for two or three days. Tincture of cocculus indicus is diluted with two to three parts of water, and this solution is used in the same way as the bichloride solution. In severe dirty cases the treatment is made much easier by cutting the hair, but this is not necessary except in the most extreme ases.

These measures kill the ova, but do not detach them from the hairs. They can be loosened and removed immediately by repeated washings with vinegar or with a lotion of acetic acid, twenty-five per cent in water. Acetic acid may be combined with bichloride in the parasiticidal lotion, and the destruction of the nits and their removal accomplished at the same time.

PEDICULOSIS PUBIS

SYNONYM: *Crabs.*

Symptomatology.—The pediculi which produce this affection locate themselves by preference in the pubic hairs. They lie close to the skin, usually clutching a hair, and often have the head partially inserted into the follicle. Their bite produces intense itching. In cleanly persons itching will likely be the only symptom, but in the careless, after the persistence of the condition for a while there will be scratched papules, excoriations, and all degrees of secondary dermatitis. But dermatitis is a less marked feature in this condition than in the two other forms of pediculosis.

The common location for this form of pediculosis is the pubic area. The parasite, however, may wander to other coarse hairs, and may be found in the hairs over the abdomen and occasionally in the axillæ, on the eyebrows and eyelashes, and on the coarse hairs of the legs. In very extreme cases they may be also found on the lanugo hairs. They very rarely affect the scalp. In a series of cases reported by Grindon the parasites invaded the scalp hairs, but only to the extent of about an inch at the border of the scalp.

Occasionally in individuals with fair, delicate skins there are seen, in connection with pediculosis pubis, so-called *maculæ cæruleæ*; that is, small pea- to finger-nail-sized steel-gray stains of the epidermis which are without elevation and do not disappear on pressure. Duguet has shown that the same pigment is contained in the thorax of the pediculus opposite the front pair of legs, where there are two salivary glands, and by producing these stains by injections of crushed crab lice has demonstrated that they are caused by pigment injected into the skin by the parasite while feeding. Maculæ cæruleæ are transitory, and disappear in a few days after the lice have been exterminated. A certain predisposition is required for the production of these spots, and they are not a constant association with the affection.

Etiology.—Pediculosis pubis is seen in all classes, and usually in adults. It is contracted by intimate contact with infected individuals and from bedding, clothing, and other infested materials. There is a very distinct susceptibility in certain individuals.

Diagnosis.—Suspicion of pediculosis pubis should be excited by the sudden appearance of intolerable itching about the genitals, especially if this is unassociated with eczema. The diagnosis has to be confirmed always by the finding of the pediculi or ova. The pediculi cling to the hairs, and as they lie close to the skin a careful search with a hand glass may be necessary to discover them. The finding of the ova attached along the hair shafts is often possible when the pediculi themselves are so few that they escape detection.

Treatment.—The best treatment for pediculosis pubis is first to wash the parts thoroughly with soap and water, preferably with corrosive sublimate or naphthol soap, and then to bathe the parts for several minutes twice daily with corrosive sublimate solution, 1: 500 or 1: 250 in water or alcohol. Ointments, such as ammoniated mercury or naphthol, five to ten per cent, or balsam of Peru and vaseline, equal parts, are efficient, but are mussy and disagreeable. The time-honored treatment by two good rubbings of mercurial ointment is nasty and dirty, is apt to excite dermatitis, and is far less effective than the use of corrosive sublimate solution. After the parasites have been killed the patient should use a soothing lotion, like calamine lotion, for a few days to relieve the irritation.

On parts other than the eyelids the treatment with corrosive sublimate can be carried out. In the eyelashes the pediculi should be picked off with forceps and the lids rubbed with dilute citrine ointment or ammoniated mercury ointment. As an additional measure to kill the nits, the lids should be sponged with carbolic-acid lotion, 1: 40. The dead nits can be removed with dilute acetic acid or vinegar, as described under pediculosis capitis.

II.—CIMEX LECTULARIUS

SYNONYMS: *Acanthia lectularia, Bedbug.*

The bedbug does not live on the skin, but comes to it to feed. It makes a puncture and, probably in order to increase the blood flow, njects an irritating fluid. The amount of irritation produced by the bite varies greatly: in some individuals there is no irritation; ordinarily there is produced an inflammatory papule or wheal, which itches inensely. There is frequently extravasation of blood at the site of puncure, producing a characteristic purpuric spot which undergoes the usual nvolution of hæmorrhage of the skin. The legs about the ankles are the site of predilection for the attack of bedbugs, probably because they are nearest the crevices of the bed where the parasite stays.

Bedbugs produce lesions which resemble urticarial wheals, but they are distinguished by the fact of their occurrence at night, the central hæmorrhagic point, and the petechiæ which characterize them.

There is rarely very extensive manifestation of scratched skin from bedbugs, and the disease does not approximate in extent pediculosis.

The treatment consists in getting rid of the parasite and in the use of soothing applications, such as calamine lotion, lotions of carbolic acid or camphor menthol, one half to one per cent; corrosive-sublimate solution, 1 : 500 or 1 : 1,000; alkaline baths or toilet vinegar; or weak antipruritic ointments, such as carbolic acid, one half per cent, or menthol or camphor chloral, one to two per cent in vaseline.

III.—PULEX IRRITANS

The common flea usually produces wheals by its bite. In some individuals there is no manifestation beyond a minute reddish point at the site of puncture. Ordinarily there is a typical pale or erythematous itching wheal with a central hæmorrhagic point of puncture. The wheals are evanescent, but are followed, especially upon scratching, by inflammatory papules which persist for several days. The itching of flea-bites can be relieved by alkaline lotions and such lotions as are suggested for bites of bedbugs. The use of camphor, menthol, or oil of eucalyptus or oil of pennyroyal affords, to a certain extent, protection from attack.

IV.—OTHER PARASITES

Gnats, mosquitoes, flies, ants by their bites produce wheallike lesions with a central puncture similar to those produced by fleas and bedbugs. The itching of the lesions is relieved by the same antipruritic applications. Some protection against their bites is obtained by the use of volatile substances, such as menthol, oil of eucalyptus, oil of cassia, oil of pennyroyal, and camphor. These can be applied in such a way as to envelop the body in the odor or can be used on the skin combined with ointments.

Various forms of **bees, spiders,** and **caterpillars** occasionally sting the skin, not for nourishment but as a defensive measure. The lesions which they produce are wheals, but they are usually large œdematous, red swellings, accompanied for a short time by painful stinging sensations. In bee stings there is usually but one lesion, the sting being broken off in the wound. Occasionally, however, as when the sting is inserted through a thin garment, the lesions may be multiple. Then they are usually less intense, and are pale, finger-nail-sized or larger wheals. The reaction produced by stings of such insects varies greatly in individuals. Occasionally they are accompanied by alarming toxic symptoms, and in rare cases death has occurred from them. The pain of these stings is relieved by bathing in strong bicarbonate of soda or other alkaline solution, or by the use of such antipruritic lotions as have already been suggested.

Ixodes or Wood Ticks.—Several varieties of ticks are temporary parasites on man as well as on various animals. Wood ticks are found on bushes and trees, and in the grass; when opportunity offers, they find lodgment on the skin, insert their probosces, and suck blood until they are full. Small at first, they may enlarge to the size of a large pea when gorged with blood. After taking their fill they fall off. Their bite produces a wheal with a central puncture, and causes more or less itching. If forcibly removed from the skin before they have finished feeding, the proboscis is likely to be broken off and cause local inflammation. They can be made to loosen by the application of turpentine or an essential oil or a little tobacco juice.

For the itching and irritation of the bite the ordinary antipruritic applications used for other parasites' bites are satisfactory.

The rôle of ticks and mosquitoes, and other animal parasites, as intermediary hosts for pathogenic organisms which are transferred to the higher animals through the bites of these parasites, is not within our scope.

Chicken Louse (SYNONYMS: *Dermanyssus avium et gallinæ, Bird Mite, Fowl Mite*).—The chicken louse occasionally attacks the human skin and produces erythematous, papular, or urticarial lesions. The parasite is pinhead sized, grayish white, and its effects are chiefly seen on the hands and forearms of those who have the care of fowls. The treatment is with the usual soothing applications.

Dermatitis from a Straw Mite.[1]—For the last several years a peculiar epidemic dermatitis has been observed in Philadelphia. Goldberger and Schamberg established that it was produced by an almost microscopic mite found in straw used in mattresses upon which the patients had slept. The mite was identified by Banks, of the U. S. Bureau of Entomology, as close to, or identical with, pediculoides ventricosus.

The affection is characterized by an intensely itching eruption of urticarial wheals, sometimes of macules or papules, most of which are surmounted by a characteristic central vesicle, which quickly becomes turbid and later pustular. These central vesicles are usually minute, not larger than a pin head; occasionally they may be as large as a pea.

The eruption appears about sixteen hours after exposure to the mite, and occurs abundantly over the trunk, and to a less extent over the arms and thighs. The itching, as a rule, subsides within twelve to thirty-six hours, and the eruption disappears in a week or ten days, although, when exposure continues, the eruption may continue for from three to seven weeks.

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[1] Goldberger and Schamberg, *U. S. Public Health Reports*, vol. xxiv, No. 28, July 9, 1909.

As a rule, there is no disturbance of the general health, but in some of the cases there is malaise, anorexia, and a temperature of 99 to 102 degrees.

As a local application for the relief of itching and the destruction of the mites on the skin the following is recommended:

Betanaphthol	ʒss;
Sulphur, precipitated	ʒi;
Benzoated lard	℥i.

The mites may be destroyed by exposing the mattresses to steam or to the fumes of sulphur or formaldehyde in a vacuum chamber.

Dermatitis from Caterpillars[1] (*Brown-Tail Moth Dermatitis*).—Various caterpillars may cause dermatitis. In England the offending species is nearly always the caterpillar of the gold-tail moth (Liparis auriflua). Within the last few years dermatitis from the caterpillar of the brown-tail moth (liparis chrysorrhœa) has become a serious nuisance in New England. The brown-tail moth is believed to have been introduced around Summerville and Cambridge, Massachusetts, in 1892, and attention was called to dermatitis from this caterpillar by Dr. J. C. White, in June, 1901. The eruption begins as erythematous macules which quickly become wheals. These are pea size, usually discrete, and in the mildest cases disappear after duration of a few hours to a few days. In the severe cases, the erythematous stage is followed by an intense dermatitis. The condition is accompanied by itching, usually intense, and in the extensive cases the general health may be greatly reduced from nervousness, loss of sleep and anorexia. The dermatitis arises from the irritation produced by the nettling hairs of the caterpillar sticking to the skin. It begins, from ten or fifteen minutes to six or eight hours after exposure, at points where the caterpillars come in contact with the skin, and there is usually a history of the patient taking one of the caterpillars from the sites of the dermatitis. These hairs also become incorporated in the caterpillars' cocoons, and the condition may be brought about from contact with the cocoons. The severity of the eruption depends in part upon the varying susceptibility of individuals, but the severer cases, as a rule, occur where the hairs have gotten widely distributed into the clothing either from cocoons or from the caterpillars. Tyzzer has shown that the reaction is not produced by the mechanical irritation of the hairs, but by a poisonous substance in them which has a strong effect upon blood corpuscles, although it does not cause hæmolysis. The poison is insoluble in and unaffected by water, alcohol, and most common solvents, and by relatively strong (one per cent) solutions of the common acids. It is soluble, however, in dilute alkaline solutions (in $\frac{1}{10}$-per-cent solution of potassium hydrate and sodium hydrate). It is unaffected by heating to a temperature of 110° C., but it is destroyed by heating to 115° C. The dermatitis is said to yield readily to mild, soothing antipruritic lotions. Washing in $\frac{1}{10}$-per-cent solution of sodium hydrate would seem to be indicated in beginning cases.

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[1] Tyzzer, *Jour. Med. Research*, March, 1907. (Review of the subject with bibliography.) Editorial, *Lancet*, Nov. 14, 1908.

Dermatitis from Minute Prickles of Vegetable or Mineral Origin.—Occasionally cases of dermatitis are produced by the mechanical injury from minute spines of vegetable or mineral origin. The eruption is an urticarial or erythematous dermatitis which is evanescent, passing off more rapidly than the lesions produced by poisonous barbs, as in the case of caterpillar dermatitis. Such dermatitis may be produced by the hairs of the fruit of cowhage (mucuna pruriens), from the silicious particles in sponges, and Wills,[1] of Bristol, has called attention to a common dermatitis of this sort occurring in stevedores handling barley imported from Northwest Africa. This dermatitis seems to come only from certain varieties of barley. The offending hairs are not the ordinary coarse barley beard, but apparently fine prickles on the main spines. It was definitely established that the hairs producing the dermatitis were of vegetable and not of animal origin.

II. DERMATOSES FROM ANIMAL PARASITES WHICH PENETRATE THE SKIN

I.—SCABIES[2]

SYNONYM: *Itch.*

Symptomatology.—Scabies is a contagious disease due to an animal parasite, the acarus scabiei, which burrows in the skin, producing peculiar lesions called cuniculi or burrows, and causing intense itching with resultant secondary dermatitis from scratching.

The acarus scabiei is a minute parasite, visible to the naked eye, from 0.3 to 0.4 mm. in length, oval, and of the general appearance indicated in the accompanying illustration. The hairlike hind legs are longer than the front legs, so that the acarus is tilted up and readily bores under the skin. The male is about two-thirds the size of the female, and does not invade the skin, but remains upon the surface.

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[1] Wills, *Brit. Jour. Derm.*, 1909, p. 249.

[2] There are various names in vogue such as prairie itch, swamp itch, lumberman's itch, elephant itch, Ohio scratches, Texas mange, and, now, Cuban itch and Philippine itch, which are used to denominate all sorts of itching dermatoses from winter pruritus to scabies and smallpox. None of these terms has any exact meaning. They are most frequently applied to scabies, but frequently also to other itching dermatoses, like dermatitis hiemalis.

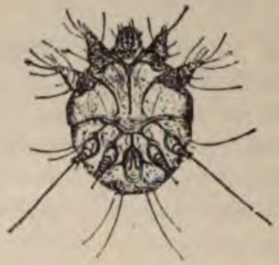

FIG. 220.—MALE ACARUS SCABEII. (Küchenmeister.)

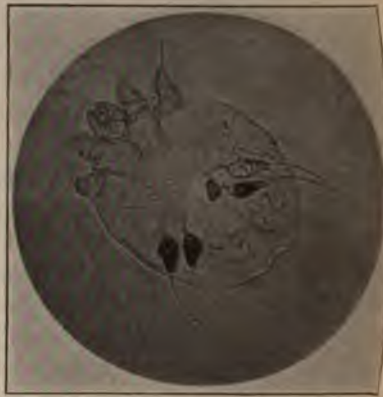

FIG. 221.—FEMALE ACARUS SCABEII. × 110. (Author's photograph of Murray Washburn's preparation.)

The symptoms of scabies are produced by the invasion of the epidermis by the female after impregnation. When placed upon the surface, the impregnated female will bore its way into the epidermis within half an hour to an hour, sometimes producing at the site a slight in-

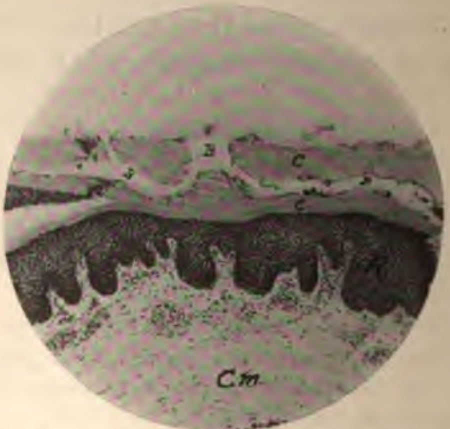

FIG. 222.—BURROW OF ACARUS. *B*, Burrow of itch mite. *C*, Horny layer of epidermis. *R*, Rete mucosum. *Cm*, Corium. (Hartzell's collection.)

flammatory reaction—a red spot or perhaps a vesicle or pustule. Once within the epidermis, the mite burrows along in the lower part of the horny epidermis, rarely invading the rete. As she proceeds she lays one or two eggs a day, and so there is produced a minute tunnel in the epidermis in which are found throughout its course ova and excrement, and at the blind end the acarus itself. The acarus lives altogether about two months, making a burrow from an eighth to half an inch in length, occasionally much longer (four inches, Kaposi). The ova develop in one to two weeks, and the larvæ are themselves capable of burrowing to a slight extent in the epidermis. They, however, reach the surface by the exfoliation of the overlying epidermis, and as this takes place first nearest the beginning of the burrow, the oldest are freed first.

The cuniculus, when it is not obscured by inflammatory reaction, is visible to the naked eye as a minute furrow in the skin. In the webs of the fingers, where the skin is thin and where they are most readily found, they show as lines about an eighth to a quarter of an inch long. The line is usually blackish, as though the horny epidermis had been scratched with a needle point without producing any inflammatory reaction and the epidermis had been uplifted slightly so that dirt became entangled in it. At the distal end of the cuniculus the acarus can often be seen on close inspection as a minute whitish speck, and can readily be caught upon the tip of a fine needle.

As can readily be understood, the burrowing of the female is followed by severe itching, and the symptoms of the disease are nearly all attributable to the scratching which is called forth. Without the secondary injuries of scratching the burrowing of the mite may produce inflammation along its track, occasionally with the formation of pustules, but this source of inflammation is relatively unimportant in comparison with the dermatitis excited by scratching.

Aside from the characteristic cuniculi—which are a very insignificant part of the clinical picture and escape observation unless carefully sought for—the symptoms of the disease are itching and dermatitis. The dermatitis is most marked at the sites of predilection, and consists usually of ordinary inflammatory lesions—discrete papular or papulo-pustular lesions surrounded by an erythematous halo. The lesions may be very abundant and grouped in the areas of predilection, but they are not likely to occur in confluent patches, such as characterize most eczemas. From secondary infection the eruption is usually of pustular character. The papules and pustules are scratched, and show abraded, excoriated tips, but there are seldom the long excoriations which characterize pediculosis corporis.

The distribution of scabies is very characteristic, and, aside from the presence of cuniculi, is the most marked feature in distinguishing it from pediculosis corporis and other forms of dermatitis chiefly excited

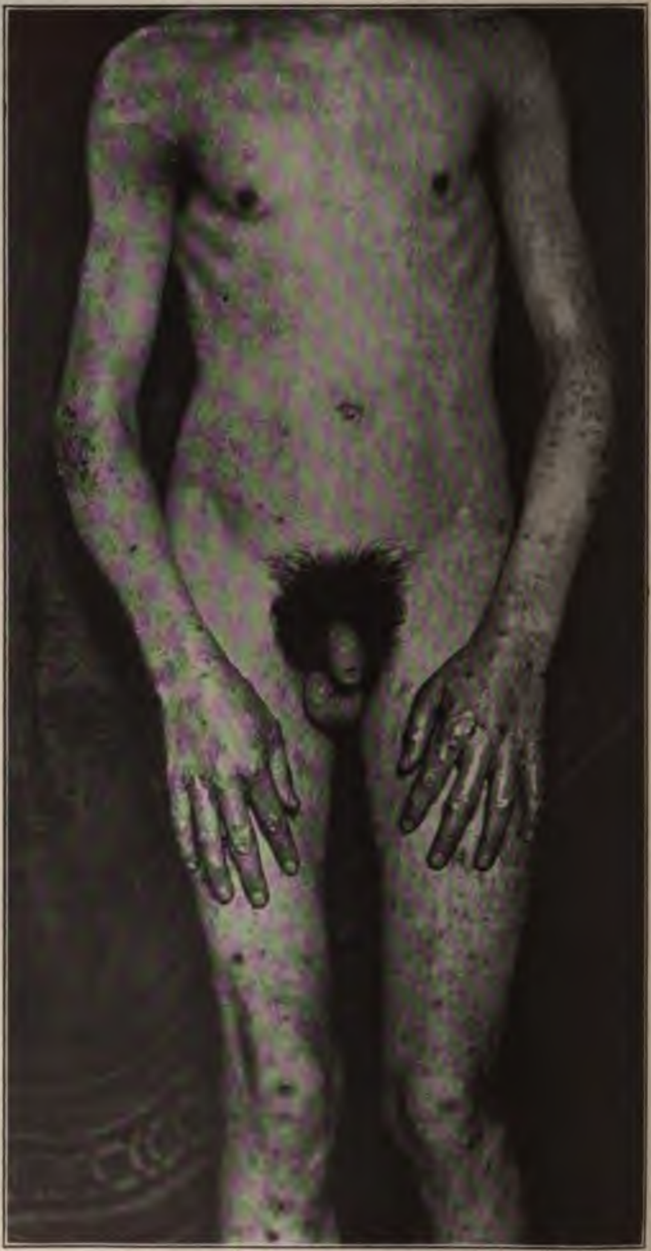

FIG. 223.—SCABIES. (Author's collection.)

y scratching. The typical locations for scabies are the webs of the ingers, the flexures of the wrists, the axillæ, the nipples, and the penis; ll surfaces where the skin is thin and where the ravages of the acarus re presumably due to the ease with which it penetrates the skin. In ddition to the webs of the fingers, the palms are usually involved; common is the involvement of the hands that the occurrence of dis-ete inflammatory lesions between the fingers and on the palms in sociation with a dermatitis on the covered parts of the body should ways suggest the suspicion of scabies. The eruption of scabies also s a predilection for parts exposed to friction and pressure, so that it is t to be marked where belts or trusses rub the surface and over the hial tuberosities in persons, like tailors and shoemakers, who at their rk sit constantly in one position.

In mild cases the lesions of scabies may be very few and be confined a few of the areas of predilection, especially the fingers and wrists, e nipples, and penis. In cases of moderate intensity the dermatitis usually most marked in its locations of predilection, but in severe ses becomes generalized as an abundant eruption over the entire body cept the face. The face nearly always escapes except in young in-nts, who contract it in that location from resting against the body their mothers. In neglected cases of very long duration scabies may oduce a picture of suppurative dermatitis of most extreme character. e surface becomes covered with a thick crust of exfoliated epithelium, s, and other inflammatory *débris* which is filled with acari. The most treme examples of this sort are found in neglected lepers, and are own as Norwegian scabies. Cases of this extreme degree are prac-ally unknown in America.

Etiology.—For the transmission of scabies intimate contact either th individuals or with infested articles is necessary. Its transmission possible by such slight contact as shaking hands or the use of towels other toilet articles, but is not likely. It is usually contracted in e intimate contact of family life, from crowding together in lodging uses and barracks, from bedding, or from infested clothing. Appar-tly it is spread among the better classes chiefly through hotels and rs. Like other verminous diseases, it is very much more common in inter, and is chiefly seen among the poor and careless classes, but abies is by no means a poor man's disease. It is frequently seen nong people of careful habits and of good surroundings. Especially this true of people like commercial travelers, whose occupations re-uire constant traveling. Unlike pediculosis corporis, it has no marked eference for the old, and occurs at all times of life.

Scabies is one of the common diseases. It has apparently in-eased greatly in frequency in the United States in the last few years.[1] At the present time it is one of the very common diseases in public practice in winter.

Diagnosis.—The characteristic features of scabies are the cuniculi and the typical distribution of the eruption. In moderate or slight cases the cuniculi can usually be found in the typical locations, especially between the fingers; in severe cases they may be entirely obscured. Their finding, especially the finding of the acarus, settles the diagnosis. But, regardless of the cuniculi, the presence of an acute papular or papulopustular dermatitis in the typical locations of scabies is almost pathognomonic of the disease. The distribution is not that of other forms of dermatitis; it differs distinctly from pediculosis corporis, with which it is most likely to be confused, in its general distribution, and especially in its involvement of the hands.

Prognosis.—Scabies yields very readily to treatment, and inasmuch as the parasite is found in the skin and not in the clothing it is a disease which can be handled much more satisfactorily in the vagrant class than pediculosis corporis.

Treatment.—The extremes which have to be avoided in treating scabies are the use of parasiticidal applications so weak that they are not effective and, on the other hand, the use of parasiticides which are so strong that they themselves produce acute dermatitis.

In the treatment of scabies it is necessary first to cleanse the skin of loose epidermis, dirt, and the products of inflammation, and to expose the acari as fully as possible. For this purpose the first measure is a warm bath with soap. If there is not too much irritation the skin should be scrubbed with a coarse washcloth or flesh brush and green soap. After this the parasiticide should be applied thoroughly at night and in the morning, and repeated for three or four days. It should be used abundantly and allowed to get into the underclothing, so that the skin is constantly exposed to it.

Sulphur is the classical remedy in the treatment of scabies and it is efficient, but it has the objection that when used strong enough to destroy the acari it is liable to set up a dermatitis of its own. It is best used, therefore, in combination with balsam of Peru, beta-naphthol, or other parasiticides. The application which I always use by preference is as follows:

℞ Precipitated sulphur,
Peruvian balsam,
Prepared chalk,
Green soap,
} āā .... gr. xx–xl;
Vaseline .... q. s., ad ℥i.

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[1] Hyde, *Amer. Jour. Med. Sci.*, 1905.

; should be applied freely at night and in the morning over all :ted areas, and should be continued as long as any evidence of the ıse remains. It is not irritating in the strength of 20 grains to ounce, and can be used vigorously. It is better to have the prenary scrub before using it, as then the applications for three days sufficient; but it can be used satisfactorily without the preliminary , although a somewhat longer time is required for cure. A somet more active (and more irritating) mixture is the following:

℞	Precip. sulphur	ʒss–i;
	Balsam of Peru	ʒss–i;
	Beta-naphthol	gr. xv–xxx;
	Vaseline q. s., ad	℥i.

Such ointments as the above usually stop the itching after one or applications, and after two to four the parasites in the skin are royed.

After the use of the parasiticidal ointment for several days the ent should have a good bath, and should then put on fresh underıing.

Julien highly recommends the method, which he first saw used in ian clinics, of painting patients with balsam of Peru without any iminary baths or other preparation. It is best applied at night, and owed in the morning or later by a bath. It usually causes no ation whatever, and is effective. It should not be forgotten, ever, that in rare instances balsam of Peru produces violent natitis.

A method which is highly recommended by Sherwell, and which is convenient, is as follows: Bathe in the evening, using an alkaline and soap over the tougher parts. Then rub the body lightly with ıed sulphur; half a dram is sufficient for one person, and hard friction ot necessary. About half a dram of washed sulphur should also be tered between the sheets of the bed, and the clothing and bed linen ıld be changed every two or three days. The amount of sulphur ssary does not produce any irritation, and cure is effected in about eek.

Precautions should always be taken to destroy the parasites which find lodgment in the underclothing and bedding. These should :horoughly boiled, and after the patient has had his bath, subsequent treatment, he should put on fresh clothing. When he has no fresh ply the underclothing should be left permeated with the parasiticides ı small quantity of washed sulphur should be dusted in his clothing, n Sherwell's treatment.

II.—SARCOPTES SCABIEI COMMUNIS

Various species of acari or sarcoptes, which are almost indistinguishable in structure and habits from the acarus that infects man, are found in the horse, sheep, dog, pig, chicken, and other animals. The female burrows in the skin in the same way as in man. These occasionally attack man and produce eruptions similar to that of human scabies, except that the burrows are not formed. These parasites, however, cannot live in the human skin, and the eruptions which they excite disappear spontaneously in six or eight weeks.

Occasionally cases are reported due to unusual acari: Pascal has described a scarlatiniform erythema in patches occurring in persons handling barley flour, and produced by an acarus which attacks the larva of a moth which infests the flour; Layet, quoted by Crocker, has reported an acarus infecting those who handle vanilla.

III.—PULEX PENETRANS

Synonyms: *Rhinochoprion, Chigoe, Chigger, Jigger, Sand Flea.*

Chiggers are minute parasites, almost invisible to the naked eye, which get upon the skin and burrow beneath the epidermis, producing inflammatory swellings, usually wheals or papules, but occasionally pustules, and even abscesses. The impregnated female alone invades the skin; it burrows into the epidermis like the acarus scabiei. The lesions are especially common about the feet and at sites where the parasite collects, as around the garters and waistband. When the parasites are numerous they may produce a very abundant eruption of inflammatory lesions and cause considerable distress from the itching. Anointing the surface with carbolized oil or kerosene relieves the irritation and kills the parasite. Oil of eucalyptus, oil of pennyroyal, menthol, camphor, and similar volatile substances offer a certain amount of protection against them.

IV.—LEPTUS AUTUMNALIS

Synonyms: *Harvest Bug, Mower's Mite.*

This is a larva of a species of trombididæ. The parasite is of brick-red color, just visible to the naked eye, from ¼ to ½ mm. in length, and of oval shape. It bores more or less completely into the skin, producing inflammatory wheals or papules, which cause violent itching and are followed by scratched skin. The parasite is active in midsummer, and is contracted from the grass and bushes, especially in swamps and on river banks. Its ravages are seen especially upon the legs and ankles, upon the hands and arms, and at times upon other parts. by the use of parasiticidal preparations, such as are used but they need not be so strong as to produce irritation, and ary scrubbing necessary in scabies is not required.

V.—DEMODEX FOLLICULORUM

: *Acarus folliculorum, Steatozoon, Entozoon, or Simonea folliculorum.*

folliculorum was discovered in the ceruminous glands by 1, and in the sebaceous glands, in 1842, by G. Simon, who t clear description of the animal.

site is microscopic in size, from $\frac{1}{8}$ to $\frac{1}{4}$ mm. in length, and c, thoracic, and abdominal segments, and of the general hown in the accompanying illustration.

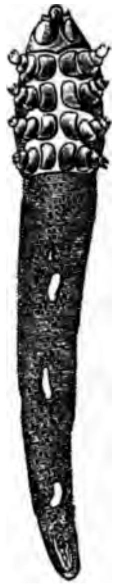

Fig. 224.—Demodex Folliculorum. (Küchenmeister.)

commonly in man, being abundant in five persons and discoverable with care ery individual except the newborn. It is abundant in people with greasy skins, large glands of the nose, forehead, and eral parasites may exist in a single folli- l it the surface should be scraped with a knife, and the scrapings mixed with glycerine and examined with a magnifi- 0 or 300 diameters. It may also be medones by teasing them in glycerine. Kraus, they are readily found by staining ith Ziehl-Neelsen stain.

of demodex is found in the dog which ppurative folliculitis, alopecia, and ca- man the presence of the parasite is un- symptoms. Occasionally, however, they tly be the cause of cutaneous symptoms. Majocchi, and Dubreuilh have reported n with abundance of the parasites, pig- f fawn or brownish color, similar to that icolor, but located on the face and neck. h's case the pigmentation was confined to areas in which was abundant. De Amicis successfully treated his case with green soap.

demonstrated a case in which the demodex was very abun- sions on the face like molluscum contagiosum, and Fordyce two cases of acne rosacea associated with a large number he sebaceous glands.

VI.—FILARIA MEDINENSIS

SYNONYMS: *Dracunculus medinensis, Dracontiasis, Guinea Worm.*

Symptomatology.—Filaria medinensis is the name applied to a nematode worm and the disease which is produced by its invasion of human tissues.

Filaria is one of the long-known diseases. It was named by Galen dracontiasis. The disease is found in various tropical countries—in India, Persia, Arabia, Upper Egypt, West Coast of Africa, and in various parts of the West Indies and South America, where it was transplanted from Africa by negroes.

Filaria medinensis is found chiefly in man, but also exceptionally in cattle, horses, dogs, cats, and other animals. Like other filariæ this parasite in its life cycle has two sets of hosts; the embryos are discharged into water, and, as shown by Fedschenko, attack fresh-water cyclops, a minute crustacean, which acts as the intermediary host in which the parasite passes the larval state. The larvæ gain entrance to the body with the cyclops in drinking water. They then escape from the cyclops, the female becomes impregnated, and wanders into the tissues, while the male is lost. It is also not positively established that the embryo may not possibly gain access to the body through the skin. The worm attains its development in the skin, and may migrate for months before it reaches the surface. Even after appearing beneath the surface it may migrate still farther before seeking exit.

The worm in the body attains an average length of two to three feet; occasionally it is much longer. It is cylindrical or slightly flattened and one fifteenth to one tenth of an inch in thickness. It is of white or yellowish color with faint transverse striations, and is firm and very extensile. In the mature worm the uterus, which is filled with innumerable embryos, is enormously developed and reaches from head to tail. In its migration it persistently descends toward the feet, the instinct which causes this, according to Manson, being directed by the fact that these are the parts which in warm climates are most likely to be brought in contact with water, where the next step in the development of the embryos takes place. In a case there is usually but one worm; occasionally there are two, and rarely they may be more numerous. When the worm reaches the surface it produces a subcutaneous tumor through the walls of which it has the feel of a mass of soft string. Over the point of exit there develops an inflammatory vesicle or nodule accompanied by some pain. This lesion ruptures, in the opening the head of the worm appears, and through it the embryos and worm are gradually extruded. The inflammatory reaction remains slight unless the worm ruptures or the head is broken off in overzealous attempts at removal and the embryos

ed in the tissues. Then violent inflammatory symptoms and even
ne, septicæmia, and death are said to be occasional results. The
development of the cutaneous lesion is, as already indicated, usu-
e feet or ankles.

mptoms during the long uncertain period before the appearance
worm in the skin are slight or absent and altogether without
teristic features. The diagnosis can only be made upon the devel-
t of the subcutaneous tumor. The characteristic features of the
are its wormlike feel, its possible change of location after appear-
nd its situation usually about the ankles and feet. The positive
sis is only to be made upon the appearance of the worm.

eatment.—The long-time native treatment is gradually to roll
rm upon a quill or small stick and thus hasten its extrusion. This
nded by the danger of breaking the worm and causing the dis-
of the embryos into the tissues. The rapidity of extrusion can
reased by douching the opening with cool water, which causes the
os to be discharged more rapidly, but even with the help of this
livery of the worm by rolling it up requires from three to ten days.
reatment now generally adopted is one which was suggested by
,[1] and has given satisfactory results in many other hands. This
s in injecting into the worm either through the opening or at vari-
ints through the unruptured skin a few drops of 1:1,000 solution
rosive sublimate. This kills both the worm and its embryos. If
rm is already extruding it can then be wound out within twenty-
ours without resistance; if not extruding it is converted into an
foreign body which, as Manson suggests, is absorbed like aseptic
.

VII.—MYIASIS[2]

iasis is the term applied to affections produced by the acci-
invasion of human tissues by the larvæ of dipterous insects. As
known, the larvæ of various dipterous insects, such as the common
e gadfly, and botfly, or œstrus, occasionally gain entrance into
in through wounds and complicate or cause inflammatory proc-
At times they obtain their entrance through the sting of the
The larvæ produce inflammatory lesions, usually furuncles, with-
culiar characteristics. Occasionally they may burrow under the
nd produce serpiginous lines of inflammation which are likely to

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rit. Med. Jour., 1894.

mith, *Inter. Med. Cong.*, London, 1881.—Dubreuilh, *Archives des médicine*
nentale, No. 2, 1894 (comprehensive review).—Abraham, *Trans. Derm. Soc.*,
ritain and Ireland, vol. iii, 1897.

end in suppuration before the larvæ escape. Their treatment consists in evacuating the larvæ and irrigating the cavities with antiseptic solutions—e. g., carbolic acid, 1 : 40.

Sting of the Screw-worm Fly

This fly occasionally attacks man. The inoculations are made by the sting of the female fly at the time she deposits her eggs in the tissue. The larvæ develop from the ova with surprising rapidity, in some cases the moving maggots being seen in the skin within a few moments after the sting is discovered. The presence of the larvæ in the tissue produces a violent inflammatory reaction with great collateral œdema, so that from a sting upon the lip the œdema may spread to the point of obstructing respiration and deglutition. Unless the larvæ are removed suppuration and necrosis occur. The symptoms are violent for a short time, but subside rapidly upon removal of the maggots or their exposure to the air. The inoculations are usually made near the nostrils, but may occur upon the lips or other parts. They are most likely to be made upon diseased surfaces, but this is not always the case.

The fly belongs to the genus compsomyia; species, chrysomya macellaria. It is a very important pest of domestic animals, but does not very frequently attack man. It is distributed throughout North and South America.

The lesions require vigorous treatment, which consists essentially in opening them and rêmoving the maggots. The very opening of the lesions tends to relieve the condition by providing for the evacuation of the maggots and by exposing them to the drying effect of the air.

An interesting case of inoculation by this fly, along with reference to the literature, is given by C. E. Tennant in the *Clinique*, October, 1906. The fly is considered at length in Bulletin No. 5 of the Bureau of Entomology of the Department of Agriculture.

VIII.—CREEPING ERUPTION[1]

Synonyms: *Larva migrans*, *Hyponomoderma* (Kaposi).

Under the names given above is described a rare lesion of the skin, produced by the wandering in the skin of a larva which leaves behind it an inflammatory track. The condition was first described by Robert Lee in 1875 and 1884, then by Crocker in 1892, and since that time sevral cases have been published. The condition must be excessively rare n the United States, although Stelwagon refers to four cases which he as seen and cases have been observed by Van Harlingen, Shelmire, and thers. In all of Stelwagon's cases the trouble began during or after a

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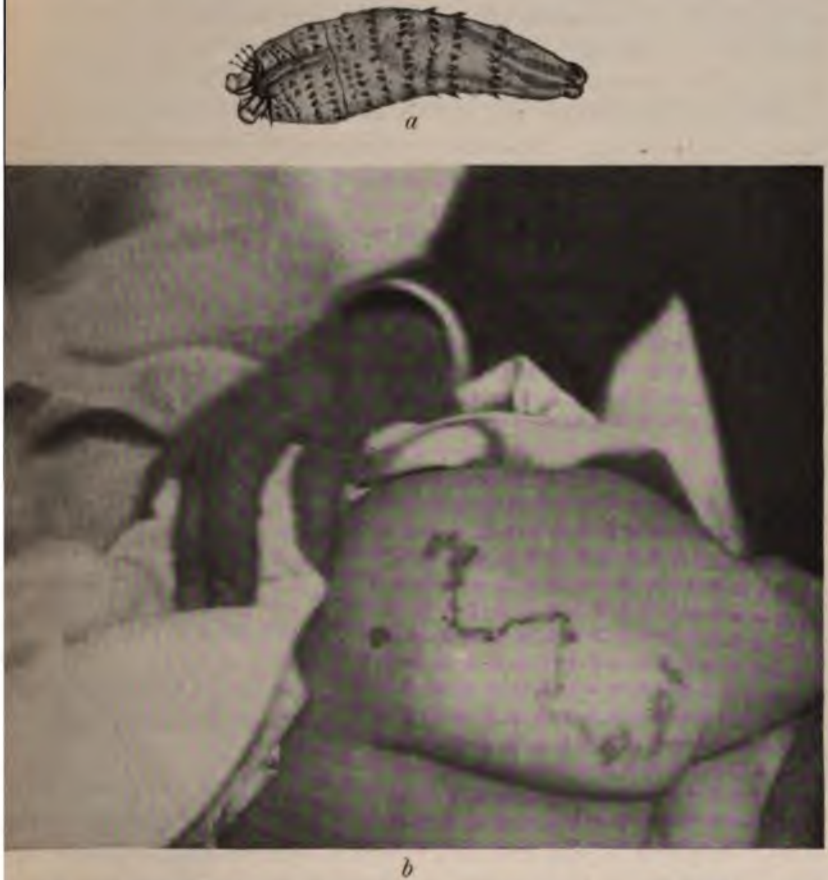
[1] Crocker's *Atlas*; *Clin. Soc. Trans.*, vol. viii, 1875; and vol. vii.—Neumann, *Archiv*, vol. xxxiv, 1896 (Abst. *Brit. Jour. Derm.*, vol. viii.—Sampson-Himmeljerna, *Abst. Amer. Jour. Cut. and G.-U. Dis.*, vol. xvi, 1898.—Hamburger, *Jour. Cutan. Dis.*, May, 1904.—Stelwagon, *Brit. Jour. Derm.*, May, 1905.—Shelmire, *Jour. Cutan. Dis.*, June, 1905.



G. 225.—(*a*) Sokolow's drawing of the Gasterophilus larva is magnified forty times. (*b*) The distinction between the fading older portion of the serpiginous eruption and the more marked active part is depicted. (Hamburger.)

journ at the seashore, so that the condition is perhaps commoner on e seacoast than in the interior. Sokoloff and Sampson-Himmelstjerna ave succeeded in recovering the larva from the skin, but others have ailed. They identify it as the larva of a dipterous insect, order œstridæ, enus gastrophilus.

The means of entrance of the larva into the skin is undetermined, ut is probably by the deposit of the ova in the skin by the insect. The larva wanders indefinitely under the skin, and produces a narrow red line one sixth to one eighth of an inch broad which is slightly raised. The line progresses at the rate of an inch or more a day, seven inches in an instance observed by Crocker. As would be expected, the direction has no regularity, but at times it may be straight. The most active part of the line is its advancing end; thence it fades out, but does not entirely disappear for several days, so that the line may be many inches in length. The larva itself is from a quarter to an inch or more in advance of the active end of the line, and its location may be indicated by some itching and burning. According to Sampson-Himmelstjerna the larva may be seen with a hand glass as a black speck by pressing the blood out of the part with a flat glass.

The duration and extent of the wanderings of this larva are indefinite. It usually goes on for several months; in a case of Crocker's it continued for two and a quarter years. While it produces a moderate inflammatory reaction it does not cause suppuration.

The rational treatment consists in destroying the larva, which is difficult because all except the Russian observers have failed to locate it. Hypodermic injections of antiseptics have usually failed to kill it. Hutchins[1] suggests first the injection of cocaine solution, followed by the injection of 1 or 2 drops of chloroform, the needle being inserted behind and pushed forward in the burrow to the probable location of the parasite.

IX.—CYSTICERCUS CELLULOSÆ CUTIS[2]

The occurrence of cysticerci of tænia solium in the subcutaneous tissues was discovered by Rokitansky. Many cases have been observed where half-cooked pork is habitually eaten.

The larvæ or cysticerci of tænia solium may invade almost any of the tissues of man. Infection is caused by the ingestion of the eggs, either with imperfectly cooked polluted food or in polluted water which is used for drinking or for washing food. Infection may also take place through contamination of the fingers with the eggs, or possibly by the passage of the eggs directly into the tissues from intestines containing tænia solium. The cysticercus is a pea-sized or larger, pale-yellowish, thin vesicle containing a colorless fluid, and within its cavity a millet-seed-sized scolex with its sucker, rostellum, and hooks projects.

When the cysticerci develop in the skin they produce oval or roundish subcutaneous tumors of normal color, usually about the size of a pea, but occasionally of greater size, up to that of a walnut. The tumors may be single, but are usually numerous and scattered. They are firm, elastic, and movable if the cysticerci are alive; after their death they hrink into hard nodules, which in the course of two or three years may ecome calcified. They may occur upon any part of the surface, but are sually found on the back and sides of the trunk, and less frequently pon the extremities.

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[1] *Jour. Cutan. Dis.*, June, 1906.

[2] Lewin, G., "Ueber Cysticercus cellulosæ in der Haut des Menschen," *Archiv*, 1894, vol. xxvi (complete review and bibliography).—Geber, Ziemssen's "Handbook of Skin Diseases."

They are painless and without inflammatory symptoms, although in are cases they suppurate.

The tumors may be mistaken for any other indolent, firm, elastic umor, and the positive diagnosis can be made only by excision or punc- re of the lesions and the discovery of the hooklets in the fluid.

The treatment is surgical removal. When the lesions are numerous, jections of bichloride solution, as used by Emily in filaria, might be fectual.

X.—ECHINOCOCCUS CUTIS

This term is applied to the rare cases of tumors in the skin produced y the larva of tænia echinococcus, the tapeworm of the dog.

Echinococcus of the skin is a rarely discovered lesion, although ac- rding to Davaine echinococcus cysts of the muscles and subcutaneous ssues occurred in 30 of 366 cases of echinococcus disease.

The echinococcus produces a soft, fluctuating, semitranslucent tumor, e size of an olive or larger, unassociated with subjective symptoms. he parasite usually dies after one or two years; then the contents of the sicle degenerate and the tumor undergoes calcification. The condition associated with hydatid cysts of other tissues. The soft, fluctuating aracter of the tumor, its superficial location and semitranslucence are ggestive points, but the diagnosis depends upon incision and the find- g of the hooklets of the parasite. The treatment is excision.

Cestode Larva in the Skin.—Stiles[1] records a case in which a pro- ferating cestode larva had been found in small tumors in the skin. he case was that of a fisherman in Florida, who presented numerous nall, acne-like tumors, from several of which Dr. H. Gates, of Manitee, lorida, recovered the worms, which he sent to Stiles for identification. he case is unique except for one similar case described by Ijimi in okio in 1905. Both the Japanese and the Florida cases occurred in shermen. Gates thinks it likely that the case is not unique along the st Florida coast.

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[1] *Jour. Cutan. Dis.*, 1908, p. 345.

Isolated or unique cases with lesions due to the accidental invasion of the skin by various other parasites have been reported.

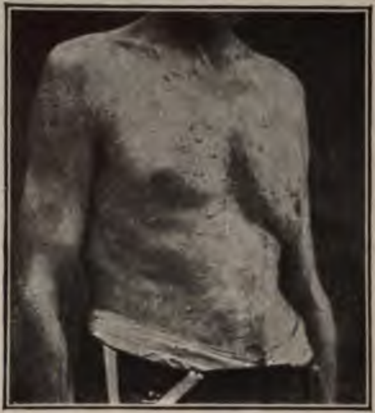

FIG. 226.—ACNE-LIKE CONDITION AND ENLARGED BREASTS DUE TO INFECTION WITH SPARGANUM PROLIFERUM. (Photograph by Gates.)

Küchenmeister has collected three cases of lesions in the skin produced by the embryo of the **distoma hepaticum.**

Sharkey has found the ova of **bilharzia hæmatobia** in skin sent him from Cairo.

Arnold,[1] of Bulawayo, found in a case of boils a larval worm a third of an inch long.

Looss,[2] in an experiment upon the skin of a leg about to be amputated, demonstrated that the larvæ of ankylostoma were able to penetrate into the subcutaneous tissue through the hair follicles. This is now a recognized route of infection.

UNCINARIASIS OF THE SKIN[3]

SYNONYMS: *Ground Itch, Toe Itch.*

This is a dermatitis due to the invasion of the skin by the larvæ of uncinaria duodenale. They produce a violent vesicular dermatitis, which becomes pustular from infection and is accompanied by great swelling and excruciating itching. The feet are the parts exposed chiefly to the invasion of these larvæ, and the lesions occur usually only on the feet, but occasionally they involve the hands. The duration of the dermatitis varies from ten days to six weeks, the longer cases being due to secondary infection from scratching. The larvæ are excreted in the feces of persons infected with uncinariasis. The larvæ are able to penetrate the skin through the follicles. C. A. Smith, who has studied the disease extensively, has shown that the dermatitis is due to an irritating substance excreted by the larvæ.

The disease is rare, but may occur whenever uncinariasis prevails. In the United States it has been observed chiefly in Georgia by Smith, of Atlanta.

Treatment is directed primarily to the relief of secondary infection. If this and scratching are controlled the dermatitis heals spontaneously

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[1] *Lancet*, April 2, 1898, p. 960. [2] *Centralblatt f. Bakt.*, May 30, 1901, p. 733.

[3] Smith, *Jour. A. M. A.*, November 24, 1906, p. 1693.

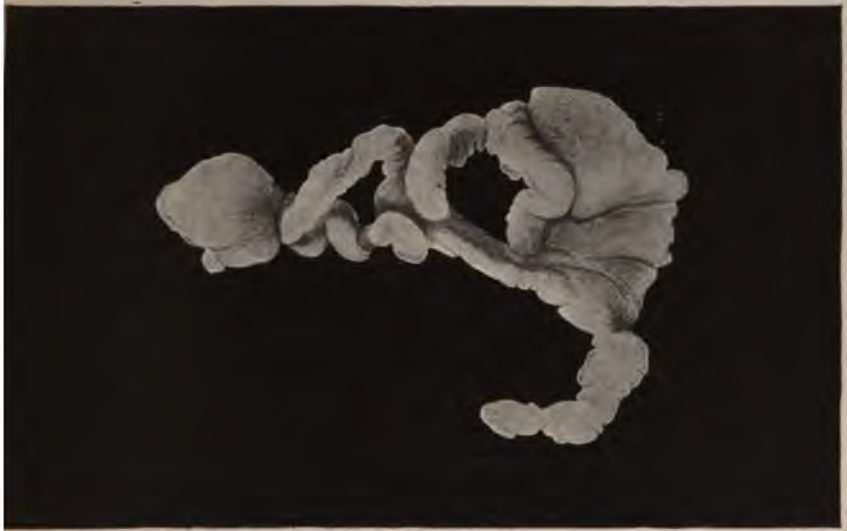

FIG. 227.—SPARGANUM PROLIFERUM, IN PART IN A CYST. × 10. (C. W. Stiles.)

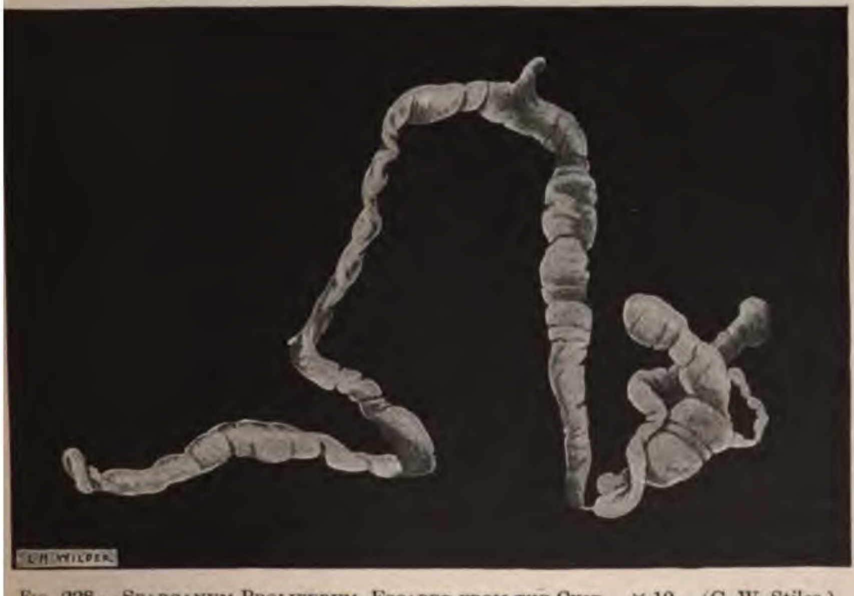

FIG. 228.—SPARGANUM PROLIFERUM, ESCAPED FROM THE CYST. × 10. (C. W. Stiles.)

in from ten to twelve days. If the eruption is seen early, it can be relieved promptly by the application of turpentine.

UNCINARIASIS OF THE SKIN.

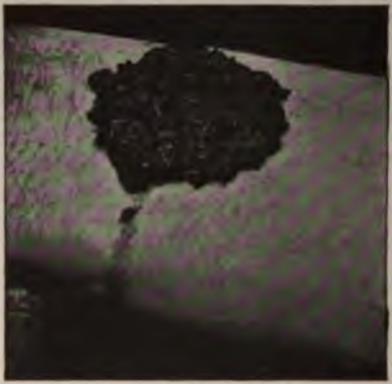

FIG. 229.—Showing application of dirt containing larvæ, to normal skin. (Smith.)

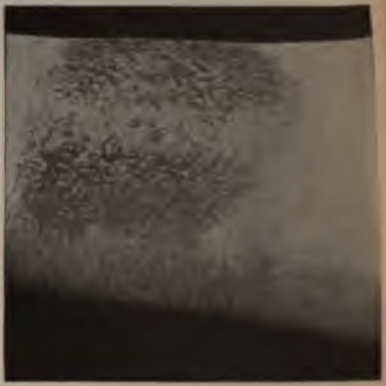

FIG. 229*a*.—Third day after applying dirt, showing vesicles at point of application. Hand swollen. (Smith.)

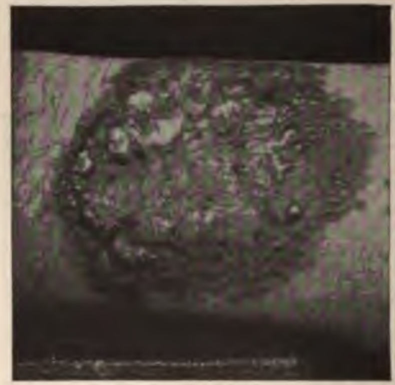

FIG. 229*b*.—Close view of eruption about fifth day. (Smith.)

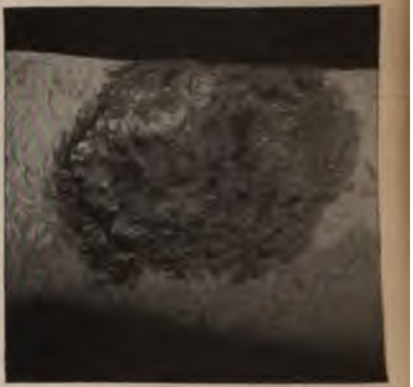

FIG. 229*c*.—Beginning scab-formation about eighth day, swelling of hand having gone down. (Smith.)

OXYURIS VERMICULARIS

Vignolo-Lutati[1] describes severe seropurulent dermatitis of the perianal and genito-crural regions produced by the oxyuris vermicularis. The patient had for some time suffered from proctitis produced by thread worms. Other cases have been recorded by Szerlecky, Michelson, and Majocchi.

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[1] *Archiv*, September, 1907.

Pruritus and secondary dermatitis from scratching not infrequently occur around the anus and vulva of children from oxyuris vermicularis. Occasionally the same conditions occur in adults.

XI.—CRAW-CRAW[1]

Craw-craw is a term which is used on the west coast of Africa to describe what are apparently various parasitic affections of the skin. A case, seen by Crocker, to which the term had been applied, was tinea cruris; others in turn have regarded it as a kind of scabies; and still other reports have associated it with filaria sanguinis hominis and with other nematode parasites.

It is probable, as Brault concludes, that the disease is not a definite clinical entity. According, however, to Emily, as quoted by Crocker, it is a definite disease with a highly characteristic course and appearance, as follows:

"Commencing as a small reddish-brown macula, situated usually on the lower extremities, but also on the dorsal aspects of the hands and elsewhere, the disease from the first is attended by an intolerable itching, which forces the sufferer to scratch himself violently. A craw-craw ulcer, when fully established, is encircled by a zone of inflamed skin of the color of wine lees, and may attain the dimensions of a five-franc piece. It consists in an excavation with nearly perpendicular sides, and a granulating bottom whence thickish pus exudes. When exposed to the air this secretion hardens, covering the surface of the sore with a dense, impermeable pellicle, beneath which the pathogenic agents, whether specific or otherwise, doubtless pullulate freely. Craw-craw has been ascribed to the gonococcus. The ulcers are invariably multiple and may occur all over the body. Dr. Spire, of Ubanghi, has met with them on the penis, where they simulated chancres, the resemblance being accentuated by inguinal adenopathy."

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[1] Emily, *Lancet*, vol. i. 1895

SECTION IX

HYPERTROPHIES

I.—VERRUCA[1]

SYNONYM: *Wart.*

WARTS are benign hypertrophic growths of the skin which anatomically show prolongation of the papillæ and hypertrophy of the overlying epidermis.

Varieties.—Both of the terms, wart and verruca (Latin, *verruca*, an elevation, a wart), are applied to several wartlike lesions, but to avoid confusion it is best to confine the terms to the common lesions which everyone knows as warts.

True warts, while essentially of one kind, are divided into several varieties because of differences in their clinical features. They all have characteristics in common: they occur most frequently upon the hands, the face, and the scalp—all exposed parts—but they may occur upon any part; their appearance and course are very erratic; they may appear suddenly without apparent cause and persist indefinitely without change; they may, on the other hand, disappear quite as suddenly and as inexplicably. They may be single, but there are usually several, and occasionally they are very numerous. The manner of their appearance, often one after another or in crops, and sometimes on opposed surfaces, strongly suggests autoinoculation. They are without subjective symptoms. They never itch spontaneously, but they are vulnerable and are easily irritated, and upon sites exposed to mechanical insult they may be tender and painful. This is especially the case with warts occurring upon the soles.

Verruca Vulgaris.—Verruca vulgaris is the common wart, with a broad base of pea size and with a rounded or slightly flattened more or s horny top. At the beginning the surface is usually smooth and thout evidence of hyperkeratosis. As the wart becomes older hyperratosis appears, the surface usually becomes roughened and papillated d of mixed blackish and grayish color.

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[1] Dubreuilh, *Annales*, 1895, p. 441; Review by Bowen, *Boston Med. and Surg. Jour.*, vol. cxxxv, 1896, p. 262.—Thin, *London Med.-Chir. Soc. Trans.*, vol. lxiv, 1881, p. 283.—Darier, *Annales*, 1888, p. 617.—Herxheimer and Marx, *Münch. med. Wochenschr.*, 1894, p. 591 (review and bibliography).—Morrow, Allen, Bronson, *Jour. Cut. Dis.*, 1899, p. 183 (discussion).—Vivès, *Jour. mal. cutan.*, 1899, p. 463.—Payne, *Brit. Jour. Derm.*, 1891, p. 184.—Jadassohn, *Verhandl. der V. Deutschen Dermatolog. Gesellsch.*, 1896, p. 497 (bibliography).—Kühnemann, *Monat.*, 1889, vol. viii, p. 341, and vol. ix, p. 17.—Fox, *Trans. Amer. Derm. Assn.* for 1888, p. 50.—Allen, *Jour. Cut. Dis.*, 1886, p. 238.

Warts occur most frequently on the hands and fingers, not infreently also upon the face and scalp, and occasionally upon any part. have seen a lesion the size of a small pea which was apparently a typil common wart, that had existed unchanged on the tip of the tongue a young man for several years. It did not show any of the changes acuminate condylomata. Warts frequently occur in the nail fold, and ay become troublesome lesions from the mechanical disturbance which ey cause. Their character in this location can be made out by the pearance of the hypertrophied papillæ which show as a group of white und points over the surface of the lesion, as in plantar warts.

Not infrequently warts occur on the soles, constituting *plantar warts* *verruca plantaris*. Here their appearance is somewhat changed by the eater pressure to which they are exposed and the greater thickness of e horny epidermis. They are flattened and have a hard, usually smooth, metimes slightly pitted surface. They have a superficial resemblance corns or small callosities, but upon closer examination their structure apparent to the naked eye; in the center the hypertrophied papillæ, sely grouped together into a bundle, show through the horny epirmis, and around them there is usually a dense horny ring. Plantar rts are usually painful on pressure, and occurring over the prominces are a source of great annoyance.

Verruca Digitata.—This term is applied to warts in which the papilry hypertrophy is much greater, and the lesions, instead of consisting of agglutinated mass of papillæ and epidermis, are a group of separated gerlike projections arising from one base. The projections may be parated down to the base of the lesion or consist of papillary excrescences ising from a warty plateau. The base may be constricted, forming a dunculated wart. They differ from common warts merely in the greater pertrophy of the papillæ. From the accumulation of dirt and horny ales the color is usually dirty grayish or blackish.

Verruca Filiformis.—A filiform wart is a threadlike wart made up one or a few hypertrophied papillæ. Filiform warts may be a quarter an inch or more in length, and vary from a sixteenth to three eighths an inch in diameter. They may be pedunculated. They are usually ft and flexible, and are seen especially about the face, neck, and eyelids.

Verruca Plana Juvenilis.—Flat juvenile warts are the usually abunnt, small, flat, smooth, glistening warts which are seen especially about e face in children. While in appearance most of their features are ke those of ordinary warts it is possible they are a distinct lesion. They are usually small, below the size of a split pea, and are flattened, only slightly elevated, of roundish or polygonal outline, and sometimes have a slight central depression, so that there may be a superficial resemblance to lesions of lichen planus. The surface is usually smooth with little or no scaliness; indeed, the lesions may be glistening and have a pseudo-translucence. Most frequently they are of the normal color of the skin or slightly grayish or brownish. They are usually abundant, appearing often in great numbers upon the faces of children, and their development, continuance, and disappearance are, as in other warts, erratic.

Verrucæ Planæ of the old are senile keratoses.

Verruca Senilis. (Cf. keratoses senilis.)

Verruca Acuminata. (Cf. condyloma acuminatum.)

Verruca Necrogenica. (Cf. tuberculosis verrucosa cutis.)

Etiology and Pathology.—Warts are commonest in childhood, but are frequent in adolescence and early adult life. There is much in their method of occurrence to suggest that they are mildly contagious. Their rapid development in great numbers, often from a "mother" wart as Vidal called it, and the occurrence, as has frequently been noted, of outbreaks of warts in individuals who are thrown in close contact, are highly suggestive of inoculation. Payne, Jadassohn, Vivès, and others have conducted more or less successful inoculation experiments. Jadassohn, by inoculations of fragments of warts from four individuals into six different persons found by development of warts within six months in thirty-three out of the seventy-four inoculations. On the whole the presumptive evidence that warts are both auto- and hetero-inoculable is strong, although no pathological organism has been discovered. Kühnemann found a bacillus which, upon inoculation into rabbits, produced suggestive lesions.

Anatomically warts consist of actually or apparently hypertrophied vascular papillæ covered by hypertrophied epidermis with marked hypertrophy of the rete (acanthosis) and with more or less hypertrophy of the horny layer. According to MacLeod, in the initial stage of verruca there is merely a thickening of the rete mucosum and stratum corneum, the underlying papillæ being flattened.[1] In the course of development all the layers of the epidermis become greatly thickened, cornification is imperfect, the cells still showing nuclei, and the horny layer dips down into the interpapillary processes, which are very greatly enlarged. The interepithelial lymphatics and the vessels of the corium are dilated.

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[1] Whether the essential change is in the epidermis or in the corium in warts is a matter of discussion. Auspitz, Unna, and Kühnemann believe that the primary process is in the rete and that the elongation of the papillæ is secondary to downgrowth of the rete.

acteria, bacilli, and cocci are found in the lesions, but that their presce is more than accidental has not been demonstrated.

The pointed warts differ from the flat ones in that they possess a cher connective-tissue core and are more vascular.

The anatomy of verruca plana juvenilis is essentially the same as at of verruca vulgaris (Thin, Kühnemann, Dubreuilh, Darier).

Diagnosis.—The appearance of warts is so familiar that there is tle possibility upon close examination of confusion with other lesions. nooth common warts may be confused with nonpigmented moles. oles are, as a rule, not so hard or of the consistence of normal skin, ow some true pigmentation, and, most important of all in diagnosis, ist unchanged from birth. Plantar warts may possibly be confused th callosities or corns. The difference in appearance has already been erred to. The flat juvenile warts suggest lichen planus, but they are normal color or slightly grayish or brownish and are quite devoid the inflammatory and other characteristic features of lichen planus. ıe flat juvenile wart might also be confused with molluscum congiosum; in molluscum contagiosum there is a central round opening ough which its contents can be expressed. For the distinction between linary warts and angiokeratoma, xanthoma, and xanthoma diabetium, with which confusion might be possible, reference is only necesy to the characteristic features of these several affections.

Treatment.—Various internal remedies are recommended for the noval of warts: nitro-hydrochloric acid, liquor arsenicalis, tincture of ıja, thyroid extract, magnesium sulphate. Magnesium sulphate sufent to produce two or three movements of the bowels a day has the hest reputation.

The only reliable treatment is local. When warts are few they may removed by destructive measures. The quickest and best method for ingle wart is to freeze it and clip it off with a pair of curved scissors, er which the base is cauterized lightly with chloride of zinc or some er strong caustic. Another method is by electrolysis, destroying the on by parallel insertions of the needle until the whole base has been terized. I have found a very good method to be sudden deep freezing h liquid carbon dioxide or liquid air. They may also be destroyed caustics. When warts are to be destroyed by caustics it is first deıble to soak off the thick horny epidermis, for this will resist almost application except the strong caustic alkalies and mineral acids. e horny epidermis can be removed by gradually dissolving with liquor assæ applied on a swab. The same thing can be done and the wart en completely removed by the daily application of salicylic acid, ʒss ʒi, in collodion ℥i. For quick removal any of the strong caustics can used—glacial acetic acid, nitric acid, chromic acid, caustic potash, zinc chloride—but these applications are not satisfactory. Daily painting with a saturated solution of potassium bichromate is recommended, but has failed more than once in my hands. One of the best local applications for many warts is the daily rubbing of the lesion with Vleminckx's solution, as recommended by Hyde and Montgomery. For the removal of large numbers of warts nothing is better than the use of x-rays. I have repeatedly had good results from a few applications of moderate intensity. Purdon[1] has had excellent results from the application of high-frequency currents.

To recapitulate: the removal of a single or a few warts is best done by freezing and snipping off with scissors, to be followed by light cauterization of the base; or by destruction by electrolysis; or by freezing with carbon dioxide or liquid air. For the treatment of many warts the use of Vleminckx's solution or of radiotherapy is best.

The treatment of plantar warts is much like that for corns. They should be pared down carefully and then covered with salicylic-acid collodion. Fitz[2] has had success from the application once or twice daily of ten-per-cent solution of chrysarobin in liquor gutta percha or in ether. If these methods are not successful the lesions must be destroyed by vigorous cauterization or excision.

II.—CALLOSITAS

SYNONYMS: *Callosity, Callus, Tyloma, Tylosis, Keratoma.*

A callosity (Latin, *callus*, hard flesh) is a thickened plaque of horny epidermis formed by hypertrophy of the horny layer as the result of continued friction or pressure.

Callosities are familiar lesions which need no description. They are seen especially about the hands and feet, but they may occur at any point which is unduly exposed to pressure or friction. They act as mechanical irritants and may be tender upon pressure. Occasionally they become inflamed and abrasions may form in which infection followed by suppuration occurs. In extreme cases ulceration may occur under callosities, and even necrosis of the underlying bone, as described in two cases, both negroes, by Crocker[3] and by Morrison.[4]

Etiology and Pathology.—Callosities are the result of an effort upon the part of the skin to protect itself against external irritation, and they are usually excited in response to friction or pressure. They may be congenital, then representing anomalies in the development of e skin. Hyperidrosis of the palms and soles is frequently an exciting ctor in their production, and they may be produced by long-continued se of arsenic, which also excites hyperidrosis.

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[1] *Dublin Jour. Med. Sci.*, vol. cviii, 1899.

[2] *Boston Med. and Surg. Jour.*, 1899.

[3] Crocker, p. 586.

[4] *Jour. Cut. Dis.*, January, 1886.

Histologically there is hypertrophy and increase in density of the orny layer. There may be thickening of the stratum granulosum. The ucous layer is, however, usually thinned and the papillæ flattened as result of the pressure; the underlying corium may or may not show e changes of inflammation.

Treatment.—Callosities, if causing no annoyance and the result of cupation, are better not treated because they furnish needed protec- on. When they are painful or otherwise troublesome their treatment much like that of corns. They should first be soaked for half an hour warm soap and water, and the surface then pared off smoothly or adually dissolved away with liquor potassæ, after which it is a good an to apply, as recommended for corns, smooth layers of adhesive aster over the surface. Hyde and Montgomery highly recommend r the removal of callosities soaking the part in warm oil, followed the application during the night of a piece of soft flannel soaked oil. Callosities may also be dissolved away by the application of xible collodion containing 30 to 60 grains of salicylic acid to the nce.

Callosities on the bottoms of the feet in patients who suffer from yperidrosis are very intractable, and are often the source of great annoy- ce, making walking a continual source of pain, and because of the tractability of hyperidrosis their treatment is very unsatisfactory. The eatment of hyperidrosis is the first consideration, after which the cal- sities may be treated in the ordinary way. The most satisfactory ethod of treating such feet is by the application of x-rays. These ould be cautiously applied from day to day until the hyperidrosis ceases. sually this occurs and the callosities disappear without the production f any x-ray reaction.

III.—CLAVUS

SYNONYM: *Corn.*

A corn or clavus (from Latin, *clavus*, a nail) is a small circum- cribed callosity which from external pressure is forced downward as conical plug.

A corn differs from another callosity only in its smaller size and onical shape, both of which are due to the fact that, occurring usually ver joints, there is pressure at one point which produces the central eep plug.

Corns are described as hard or soft. The appearance of hard corns familiar. They occur over bony prominences, especially on the toes.

Soft corns occur on the lateral surfaces of the toes, rarely of the fingers, as a result of pressure or friction of the adjacent parts. They differ in appearance from the hard corns because, on account of the maceration to which they are constantly exposed, the horny plug remains yellow and softish. Corns on pressure may be exquisitely painful. They are usually more or less tender and are frequently weather-sensitive, pain occurring in association with storms. They may become inflamed and even ulcerate. Inflammation and suppuration are not uncommon around soft corns.

Anatomically corns consist of dense conical masses of horny epidermis. The papillæ beneath them are obliterated and those around are hypertrophied. According to Rindfleisch corns are preceded by hyperæmia, which is the cause of the hyperplasia of the horny layer.

Treatment.—To remove a corn the foot should first be soaked for fifteen to thirty minutes in hot water with soap, after which the corn should be pared off smoothly. A skillful chiropodist can dissect out the "core" without producing bleeding, but this is not a safe proceeding with the dirty knives that they use. In the ordinary paring of corns care should be taken not to cut into the corium of the toe, and the knife should first be washed with alcohol. After paring, the corn may be protected from pressure by the wearing of a felt ring which must be properly applied and changed every day. A better plan, recommended by Hyde and Montgomery, is to cover both the corn and the adjacent skin with narrow, short, smoothly adjusted strips of adhesive plaster; or instead of this Burgundy pitch may be melted and painted over the surface. Instead of paring a corn it may be dissolved away by carefully rubbing with liquor potassæ on a small cotton swab. Another method of removing corns which is useful when they are tender is by the application of salicylic-acid mixtures. The following is a common and good formula:

℞	Salicylic acid	gr. xv–xxx;
	Ext. cannabis indica	gr. x;
	Flexible collodion	℥ss.

To be carefully applied over the corn only, and to be replaced as often as it wears off until the plug separates or irritation is produced.

Soft corns should be kept thoroughly dry and clean. They should be dusted with boric acid or some inert powder, and the parts separated by a pledget of cotton. This treatment is usually sufficient for their removal, but when necessary they may be pared carefully before this treatment, or they may be treated by the application of salicylic-acid collodion.

IV.—SYMMETRICAL KERATODERMIA OF THE EXTREMITIES[1]

ONYMS: *Keratodermia palmaris et plantaris, Congenital or Hereditary Keratoma of the Palms and Soles, Ichthyosis palmaris et plantaris, Keratodermia symmetrica.*

is term is applied to rare cases exhibiting symmetrical and diffuse
of hyperkeratosis on the palms and soles, which are essentially
hic origin and are only in part, if at all, the result of external
ical influences.

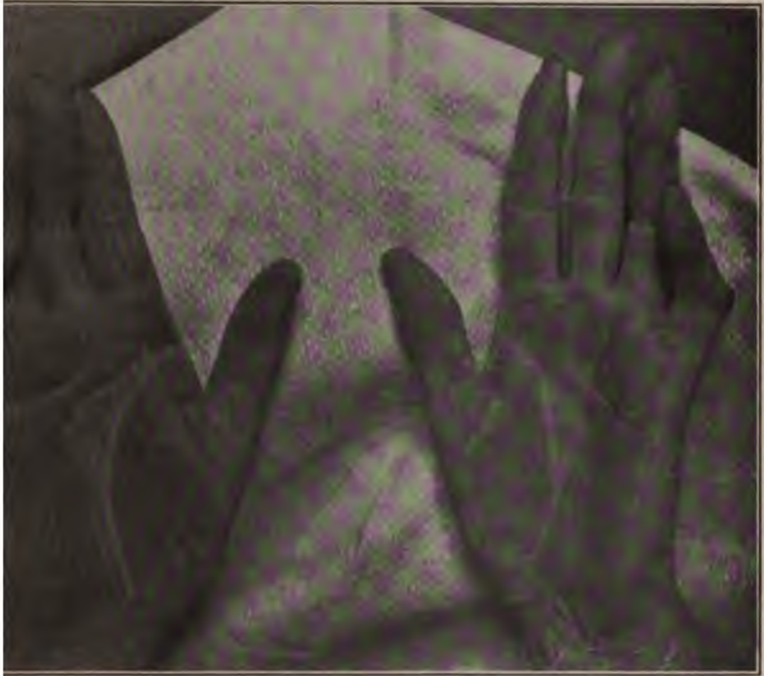

FIG. 230.—SYMMETRICAL KERATODERMIA. (Author's collection.)

mptomatology.—The affection consists essentially of great thick-
of the horny epidermis which forms plates from the thickness
nger nail up to an eighth of an inch or more. When seen in its

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nna, *Archiv,* 1883, p. 231.—Hyde, *Med. News,* vol. li, 1887, p. 416 (bibli-
); Morrow's "System," vol. iii, p. 405.—Discussion, *Trans. Third Internat.*
l. Congress, London, 1896.—Crocker, *Brit. Jour. Derm.,* 1891, p. 169.—Besnier,
Atlas Rare Skin Dis., 1889, Plate V, "Keratodermia symmetrica erythe-
"—Brooke, *Brit. Jour. Derm.,* 1891, p. 335.—Sherwell, *Jour. Cut. Dis.,* 1898,
-Neumann, *Archiv,* 1898, vol. xlii, p. 163.—Pendred, *Brit. Med. Jour.,* 1898.
r, *Archiv,* 1901, vol. lvi, p. 1.

earliest stage the hyperkeratosis is usually in the form of papules around the follicles of the skin, or it may be in a thin diffuse layer; in either case the finer markings of the palms and soles are apt to be obliterated, while the coarser markings are deepened. The well-developed patches may be quite smooth, or rough and pitted. In extreme cases the thickest plates, which are usually found on the heels, may be a quarter of an inch or more in thickness with smooth or roughened surface and an irregular blackish horny border. The condition, when once fully established, is usually permanent. Large plates may be occasionally thrown off, but quickly re-form.

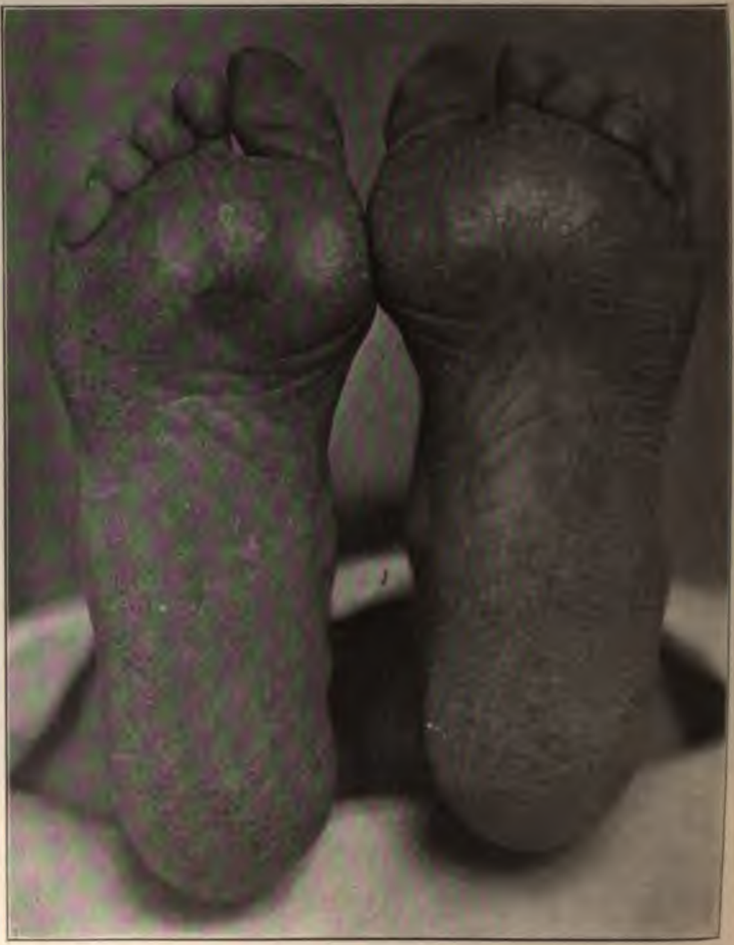

FIG. 231.—SYMMETRICAL KERATODERMIA. (Author's collection.)

Over the palms the hyperkeratosis is usually uniform. On the soles is most marked on the heel and ball of the foot, while the arch escapes shows the hyperkeratosis to a much slighter degree. The plates may tend beyond the palmar and plantar surfaces, and may be associated th hyperkeratoses on the knuckles and elsewhere on the dorsal sur-es. Brocq has described a case in which the hyperkeratoses were in eaks parallel to the bones along the anterior surfaces of the fingers. ie borders of the plates are usually sharply defined and without in-mmatory areola. Occasionally, however, there is an erythematous der around the plates, and on the soles in greatly thickened cases the lammation at the base and around the borders may be pronounced. such cases painful fissures are likely to occur.

Cases distinctly characterized by an erythematous halo, which is arently quite independent of the mechanical irritation of the horny sses, have been described by Besnier as *keratodermia symmetrica ery-matosa* and by Brooke as *erythema keratodes of palms and soles.*

The lesions of symmetrical keratodermia are insensitive in them-ves, but from the mechanical disturbance which they cause, particu-ly on the soles, the parts may become very tender and walking be de painful. In an extreme case under my observation walking beyond ew steps was impossible.

Mal de Meleda.[1]—Symmetrical keratodermia of the extremities is lemic on the island of Meleda, off the coast of Dalmatia, and has n suspected there to be a manifestation of leprosy. Neumann and iers who have examined some of the cases found them to be sym-trical keratodermia, apparently identical with the cases occurring ewhere and showing no manifestations of leprosy.

Etiology and Pathology.—The condition may be congenital or uired. In both the congenital and acquired cases heredity is an im-rtant factor in the etiology. Groups of cases occurring in many mem-s of the same family (sixteen cases in a family of forty people) and several (four) generations have been reported by Vörner, Brayton, croo, Pasini, Pendred, and others.

In both the congenital and acquired cases hyperidrosis is an impor-nt predisposing factor. In rare cases the condition is the result of the ng-continued use of arsenic, which also produces hyperidrosis of the tremities. The association of the condition with pemphigus, dermatitis rpetiformis, psoriasis, and other diseases, in which arsenic is com-only given, is probably attributable to the arsenic. Hyde has found e condition associated with slow pulse in individuals in apparently good health, and some of the cases have been in patients with various nervous disturbances.

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[1] Neumann, *Archiv*, 1898, vol. xlii, p. 163.—Hovorka, *ibid.*, 1896, vol. xxxiv, 51.—Hovorka and Ehlers, *ibid.*, 1897, vol. xl., p. 251.

The condition is a trophic disturbance probably due to various causes. The histological changes are those of callosities.

Diagnosis.—The occurrence of symmetrical keratotic plaques, often on both palms and soles, more diffuse and of more exaggerated type than those resulting from pressure and friction, often congenital or hereditary, furnishes a syndrome which is characteristic.

Treatment.—The treatment is similar to that for other callosities. The use of x-rays offers some prospect of radical relief. Other treatment is only palliative.

V.—KERATOSIS SENILIS[1]

SYNONYMS: *Seborrhœic Keratosis, Senile Keratoma, Senile or Seborrhœic Warts Senile Seborrhœa, Acné sebacée partielle, Acné concrète, Verruca plana.*

Senile keratoses are the flat, more or less roughened and scaly keratotic patches which occur in the aged.

They are very common lesions on the faces of old people. They are one of the manifestations of the degenerative changes in the skin which occur in old age, and should be considered in connection with senile atrophy of the skin. They are of importance because of the frequency with which they develop into epitheliomata.

Symptomatology.—Senile keratoses begin as circumscribed brown pigmented spots in the skin. Such spots appear irregularly without apparent cause and change very slowly. Occasionally such a spot will thicken and become slightly elevated and persist for years as a smooth, flat, brownish plaque from the size of a wheat grain to a finger nail, and which closely resembles a slightly elevated, flat, light-brown mole. As a rule, however, after years the patches become more or less roughened and covered with closely adherent small but thick scales. The appearance of the fully developed patches varies according to the degree of hyperkeratosis. They may be hardly appreciably elevated, perhaps slightly greasy, and of yellowish-brown or grayish color. In their extreme development they are covered by firm, dry, grayish or blackish scales of the thickness of a finger nail. There is bleeding upon removal of the scales, and gaping follicles may be exposed, with corresponding spines on the under surface of the scales.

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[1] Hartzell, *Amer. Jour. Med. Sci.*, 1899; *Jour. A. M. A.*, April 16, 1898; *Jour. Cut. Dis.*, September, 1903.—Dubreuilh, *Annales*, Tome vii, Troisième Série, 1896, p. 1180; *Trans. Internat. Dermat. Congress*, 1896.—Montgomery, D. W., *Phila. Med. Jour.*, 1898, p. 211.—White, R. P., *Brit. Jour. Derm.*, January, 1903.—Watson, *ibid.*, January, 1904.—White, J. C., *Boston Med. and Surg. Jour.*, vol. cvii, 1882, "The Old Age of the Skin."—Pollitzer, *Brit. Jour. Derm.*, 1890, p. 199.

The patches are irregular in outline and usually from the size of
ckhead to a finger nail; occasionally they may be as large as a small
. Their typical location is the face, especially the cheeks and nose.
y occur very frequently on the backs of the hands, and they also at
es develop upon covered parts, especially the chest and shoulders.

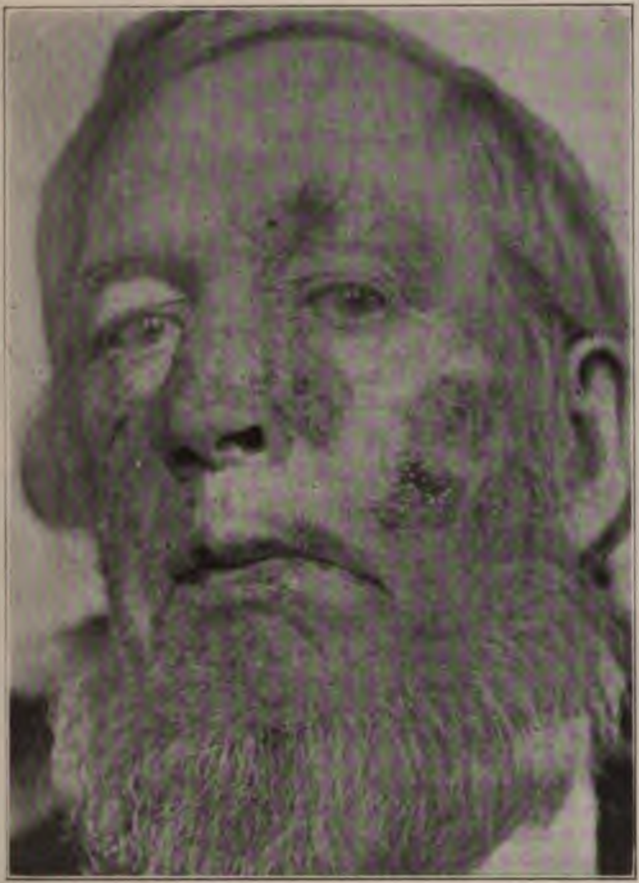

FIG. 232.—SENILE KERATOSES—SOME OF THEM EPITHELIOMATOUS.
(Author's collection.)

ir entire number varies greatly; usually there are several of them in
rent stages of development. There may be only one or two, or they
be so numerous on the face and hands as to give the parts as a whole
ughened, scaly appearance. Frequently around them there are many
ted capillaries, and in the cases where the lesions are numerous all
he skin of the face and of the hands, if they are involved, is usually
d, slightly scaly, and roughened. In association with the lesions

there is often dry seborrhœa or seborrhœic dermatitis of the scalp and ears, and less frequently also of the face.

The lesions may persist indefinitely without showing any evidence of inflammation underneath them, but usually sooner or later, particularly in the abundant cases, the bases of some of the keratoses become slightly inflamed and show a red border. When this occurs the lesions have already become epitheliomatous, and the underlying epithelium shows the structure of cutaneous carcinoma. Not infrequently one sees cases where there are many small epitheliomata in varying stages of development along with the senile keratoses. The epitheliomata which accompany them are usually very indolent and of very slow growth. They are,

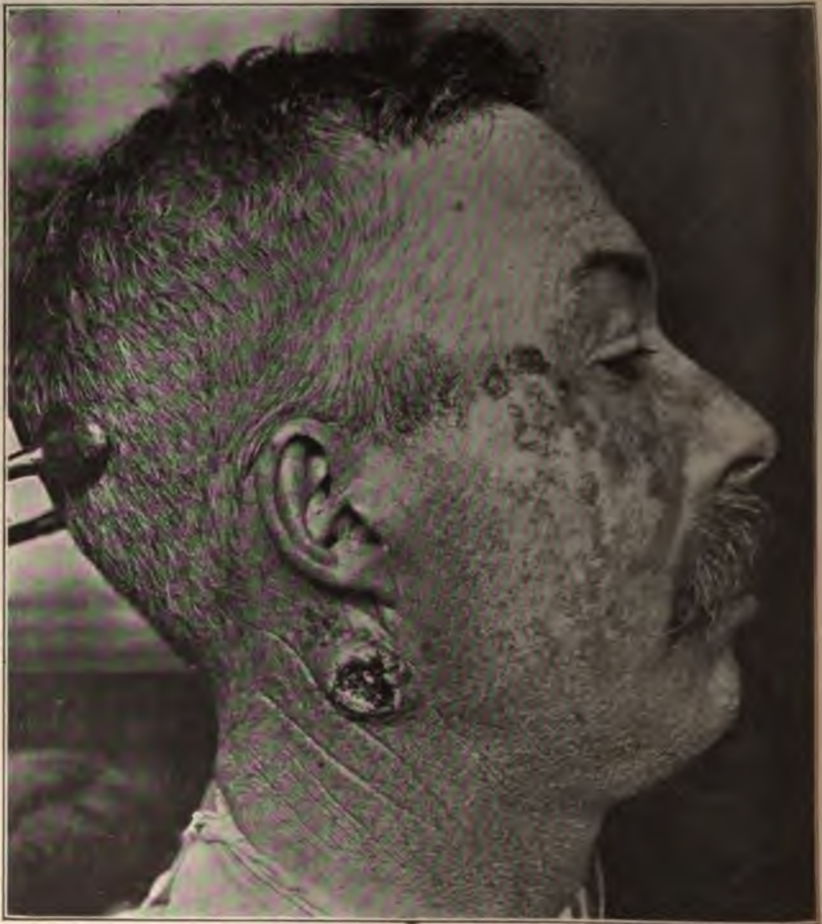

Fig. 233.—Senile Keratosis with one Craterform Epithelioma. (Author's collection.)

perhaps, most frequently flat and superficial, of the clinical type of rodent ulcers, but very often the epitheliomata are nodular although superficial.

The keratoses are without subjective symptoms.

Etiology and Pathology.—The most important factor in the production of senile keratoses is the degenerative changes in the skin which produce the condition recognized as the senile skin. Senile keratoses are accordingly seen, as a rule, in the old, but they may occur early in life; I have seen a well-marked example of the condition in a man of twenty-seven. The most important factor, in my opinion, in the production of senile keratoses is a peculiar quality in the skin. The abundant cases occur in a peculiar complexion—in individuals who are florid, with many dilated capillaries coursing through the skin, with the skin itself harsh and dry and showing more or less evidence of dry seborrhœa. The condition is not infrequently in my experience seen in more than one member of the same family.

J. C. White has pointed out that the condition is likely to develop in persons who are not careful in keeping the skin free from accumulation of sebaceous secretion. I should say rather that the condition is likely to occur in patients who have a persistent dry seborrhœa which renders it impossible to keep it free from accumulation of greasy scales. The condition undoubtedly occurs in individuals who have habitually been cleanly.

Next to personal idiosyncrasy and age, the most important factor in the production of senile keratoses is a mode of life which involves constant exposure to sunlight and wind, and the lesions are seen most frequently in coachmen, farmers, and other persons in exposed occupations.

Hartzell has recently studied the histology of the subject. He found great thickening of the horny layer with persistence of the nuclei of the cells as well-stained, slender spindles. There was great thickness of the horny layer about the mouths of the hair follicles and sweat glands, and the mouths of the hair follicles were dilated and filled with horny plugs. The granular layer had disappeared except about the openings of the hair follicles and sweat ducts. In the rete in the earlier lesions there was slight increase in the thickness of the layer with evidence of increased activity in the basal cells. “ In the older lesions there were all degrees of increase in the thickness of the rete up to actual invasion of the corium and beginning epithelioma.” Contrary to the findings of some other students, there were no changes in the sebaceous glands. In all of the cases changes were found in the sweat glands. There was proliferation of the lining epithelium with increase in the size of the cells, causing obliteration of the sweat tubules at some points with cystic dilatation at others. Cellular exudate was frequent around the glands, The corium presented nothing abnormal except flattening and, in some cases, disappearance of the papillæ.

Diagnosis.—The lesions are gray, scaly, flat, wartlike patches on the hands and face of old people or of those showing evidences of senile changes in the skin; the patches are often upon inflammatory bases, some of them perhaps already epitheliomatous; the lesions occur in a greasy or harsh, often florid, skin; quite frequently there are accumulations of sebaceous scales. This complex of symptoms is characteristic and cannot easily be confused with anything else.

Prognosis and Treatment.—The ordinary course of the lesions is slowly but persistently to increase. They do not, as a rule, disappear spontaneously. After the patches have persisted for years some of them are apt to develop into epitheliomata, which, however, usually pursue a slow course and may, on account of the age of the patients at which they develop, cause no serious trouble.

The condition may be greatly improved by the free use of soap and water, combined with daily greasing of the skin with a bland fat, like lard, or rose ointment, or vaseline. An improvement over the simple ointment is made by the addition of 5 to 20 grains of both salicylic acid and sulphur to the ounce of ointment. The face should be washed at night with soap and water, thoroughly but not so vigorously as to irritate it, after which the ointment should be applied. It may be necessary to repeat the washing and greasing of the skin twice daily. By these measures great improvement can be gotten in the keratoses. They may apparently disappear, but when care is relaxed they recur. If the lesions have become inflammatory these measures will only improve them, not cause their entire disappearance.

Strong applications of salicylic acid and other keratolytic agents are sametimes advised, but from the already present danger of epithelioma irritating agents should not, in my opinion, be used unless they are to be applied to small areas and in sufficient strength to act as escharotics and destroy the bases of the lesions.

Senile keratoses, either before or after they have become epitheliomatous, can be radically removed with little or no scarring by the use of x-rays. When the patches are few the applications may be made to each patch individually, and cautiously carried along from week to week until the patches disappear or slight erythema is produced. Upon the production of erythema the patches usually promptly disappear and are succeeded by smooth normal skin. When the lesions are numerous either side of the face or the back of either hand can be exposed at once. Before erythema occurs the lesions usually disappear or so greatly improve that further treatment is not needed. My experience in a considerable number of cases treated with x-rays has been eminently satisfactory.

re is some tendency for the lesions gradually to reappear, but the lition, as it were, begins *de novo* again. Improvement has remained ome of my cases for as much as four years.

VI.—CUTANEOUS HORNS[1]

SYNONYM: *Cornu cutaneum.*

Cutaneous horns are hypertrophic masses of horny epithelium which me hornlike shapes. They are rather rare lesions.

Symptomatology.—In appearance they resemble horns in minia-. They are usually curved or twisted, and their surface is rough or inated and of a grayish or blackish color. Ordinarily they are small, diameter of an eighth of an inch or more and a quarter of an inch

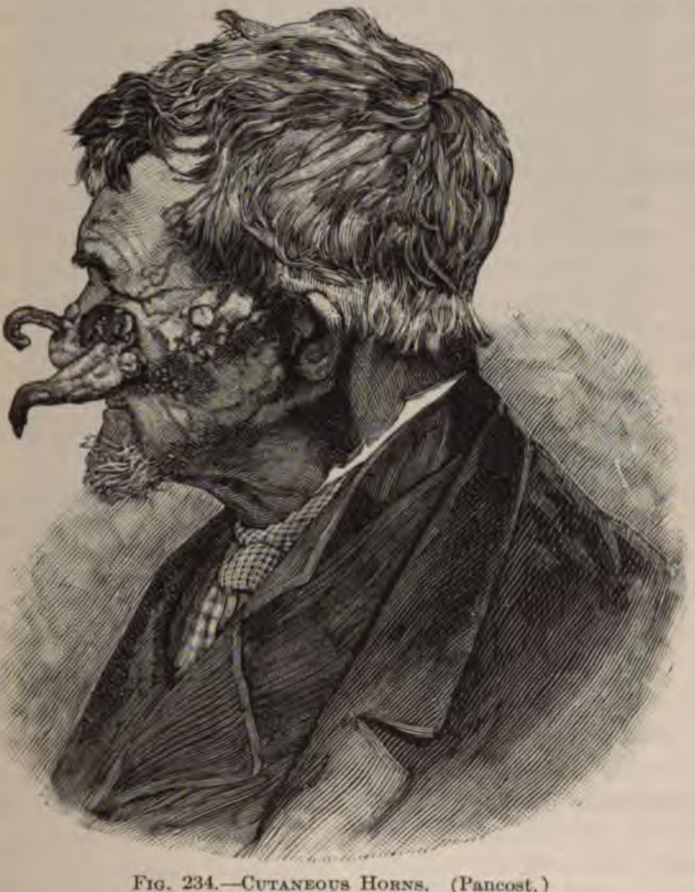

FIG. 234.—CUTANEOUS HORNS. (Pancoast.)

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[1] Brinton, *Med. News*, August 6, 1887.—Marcuse, *Archiv*, 1902, lx, p. 197.

or more in height. Extreme examples have been seen which were four or five inches in diameter and others which were twelve or fourteen inches in length. Their attachment is firm and they can only be removed by force, but they may be spontaneously shed. The surrounding skin may be normal or inflamed. Sometimes suppuration at the base occurs, and this may result in throwing off of the horn. They are usually single, but may be multiple and in large numbers. Their commonest location is on the face and scalp, but they may occur upon any part. Fifteen cases are recorded in which the lesions were situated on the penis. Lewin and Heller[1] have described palmar cutaneous horns 2 cm. in height developing upon palmar syphilides. They usually develop very slowly and after reaching a certain size remain stationary, but occasionally their growth is rapid. After persisting for a longer or shorter time it is not uncommon for the base to degenerate into epithelioma. According to Lebert this occurs in twelve per cent of the cases. My impression is that this change is much more frequent.

Etiology and Pathology.—Cutaneous horns have been seen in the young, but they usually occur in old age along with senile keratoses. Most frequently, when not arising from senile keratoses, they start in horny warts; they are in fact often, as Hyde points out, hypertrophic horny warts. They may also begin in scars, sebaceous or fatty cysts, and in other lesions.

The base of a horn usually consists of hypertrophied papillæ, which form a rounded projecting mass over which a cup-shaped depression in the horn fits. In the corium there are usually dilated blood vessels and more or less evidence of inflammation.

Treatment.—Treatment consists in removal of the horn and destruction of the base. This may be done at once by excision; or the horn may be dissolved off with solution of caustic potash, or forcibly broken off and the base cauterized with caustic potash or chloride of zinc.

VII.—ANGIOKERATOMA[2]

SYNONYMS: *Keratoangioma, Lymphangiectasis, Verrues télangiectasiques.*

Angiokeratoma is an affection characterized by the development of telangiectases in the skin upon which wartlike hyperkeratoses develop.

Symptomatology.—The name angiokeratoma, given to the affection by Mibelli, well describes the lesion. The underlying and apparently essential feature of the process is permanent dilatation of superficial capillaries in the skin. The condition first manifests itself after chilblains or some other disturbance of peripheral circulation, as pinpoint pinhead size, discrete but closely crowded telangiectases, which may pink or purplish, according to the amount of peripheral congestion. ater hyperkeratosis takes place over the telangiectases and the lesions come wartlike. Adjacent minute lesions may coalesce, forming a lesion

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[1] "International Atlas Rare Skin Diseases," Part VII.

[2] Fordyce, *Jour. Cutan. Dis.*, March, 1896.

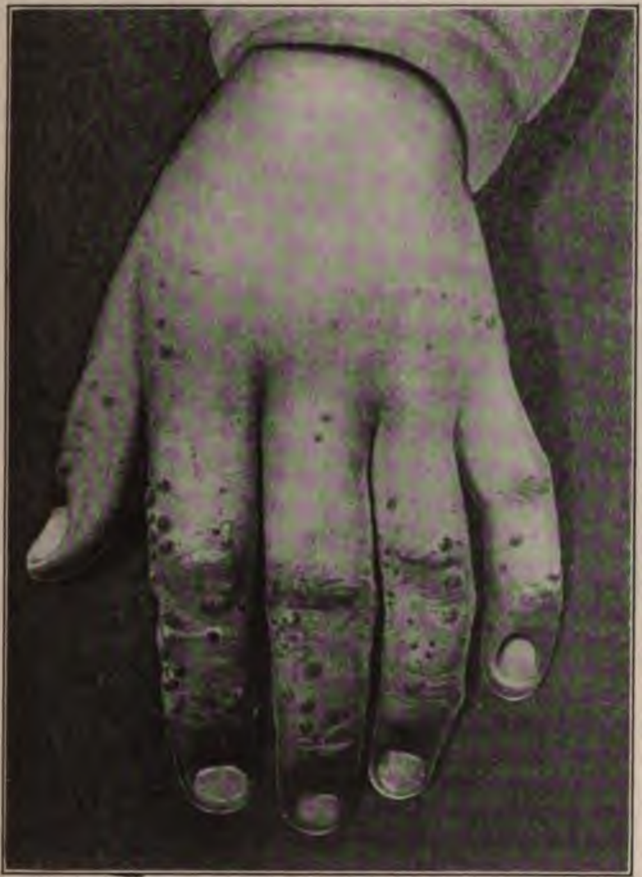

FIG. 235.—ANGIOKERATOMA. (Photographic reproduction from Mibelli's colored plate, International Atlas.)

raction of an inch in diameter, covered with irregular horny projecns. The lesions are usually abundant on the affected parts, and are inarily to be found in different stages of evolution. The most frent locations of the lesions are in terminal areas of circulation, espelly the dorsal surfaces of the fingers and toes. They also occur on palmar surfaces, and may be found upon various other parts of the ly. In Fordyce's case they were on the scrotum. In Zeisler's case re were lesions on the hands, feet, ears, and forearms; on the legs, doubtless as a result of the favorable influence of gravity in causing greater dilatation of the telangiectases, there were pedunculated vascular tumors and nævuslike lesions.

The lesions show no tendency to spontaneous disappearance and the disease gradually becomes more marked. So far as I know there are no reported cases of epithelioma developing in them as might perhaps be feared in old age.

There are no subjective symptoms.

Etiology and Pathology.—The disease is rare. In most of the cases the disease has begun in childhood, although Zeisler's and Fordyce's patients were both beyond middle life at the time it appeared. Some of the reported cases indicate a hereditary tendency. The important factor in the production of the condition is vascular disturbance tending to peripheral congestion, and most of the cases have followed chilblains.

Most observers are agreed that the primary pathological change is vascular dilatation with secondary hyperkeratosis. Microscopically there are found cavernous spaces in the derma just beneath the rete. Over some of these dilatations in the derma, spaces filled with blood may be found in the rete itself. Above the cavernous spaces there is hyperplasia of the rete and thickening of the horny layer.

Treatment.—As Pringle has suggested, the lesions of angiokeratoma may be destroyed, like other telangiectases, by electrolysis. A needle attached to the negative pole must be inserted into the base of each lesion successively, and a current of three to five milliampères passed for a few seconds. Measures should be taken, as in chilblains, to furnish proper protection to the affected parts against cold.

VIII.—POROKERATOSIS[1] (Mibelli)

Synonyms: *Hyperkeratosis eccentrica, Keratodermia eccentrica, Hyperkeratosis figurata centrifuga atrophicans.*

A disease characterized by the presence of one or more slowly spreading patches which consist of a horny ridge at the border with a less elevated or depressed central area.

The disease was described simultaneously in 1893 by Mibelli and Respighi, and cases have been reported subsequently by Hutchins, Reisner, Joseph, Wende, Gilchrist, and Basch.

Symptomatology.—In Wende's case the disease began in what was apparently an ordinary rough scaly pea-sized wart, which after a year suddenly changed into a small ring which spread peripherally. The lesions e coin size or larger, without evidence of inflammation, and consist of e horny ridge, which forms the border, surrounding an apparently or lly atrophic center. In the well-developed patches the horny ridge sharply elevated and well defined, and it may be grooved on the top a sulcus, which may in turn have a horny thread dividing it into latl halves. The ridged border may be broken or continuous. Upon the rface of the ridge there may be found minute blackish horny concrens which can be ked out, or papry horny projecns like minute rts. These may be found occanally in the insed area. In illined patches the der may be sim- a loose rim of idermis loosened d slightly uplift- on the inner side. he central portion the lesion may normal or atrohic. The horny idermis is thicked and may be scaly or smooth. There may be disappearance of the irs, and there is diminution or absence of the secretion of sweat and bum in the affected areas. The color of the border is dirty grayish or ackish, while that of the center is of a lighter shade of gray or, in rly lesions, it may be perhaps pinkish.

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[1] Mibelli, *Giorn. ital.*, 1893, p. 313.—Respighi, *ibid.*, 1893, p. 356.—Hutchins, *Jour. Cut. Dis.*, 1896, p. 373.—Wende, *ibid.*, 1898, p. 505.—Gilchrist, *ibid.*, 1899, p. 149.—Heidingsfeld, *ibid.*, January, 1905 (review and bibliography).

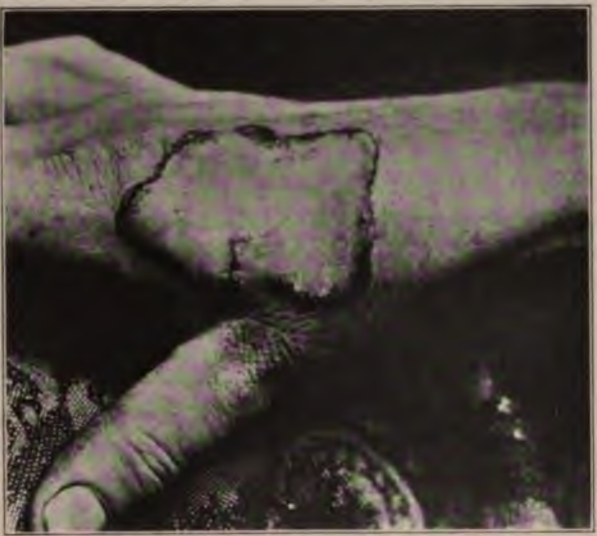
FIG. 236.—POROKERATOSIS. (Grover W. Wende's collection).

There may be a single patch or the patches may be numerous in varis stages of evolution and widely distributed. The patches may be undish, square, or of irregular outline. In size they vary from half inch to several inches in diameter, and the confluence of adjacent tches may involve larger areas, the whole of the forearm, for example. a rule, they gradually spread, but they may after a certain length time remain stationary. The condition improves in warm weather d becomes worse in cold. Involution rarely occurs.

The sites of predilection are the dorsal aspects of the hands and feet. ey may occur on the palms and soles, and have been seen on the face, e limbs, and the trunk. In three of their four cases Respighi and ucrey have also found lesions on the mucous membranes, showing as irregular, opalescent, spreading patches inclosed by a raised line around which there was a zone of hyperæmia.

There has been variable itching in some of the lesions observed.

Etiology and Pathology.—The disease is excessively rare. Cases have been reported in this country by Hutchins, Wende, and Gilchrist. It is apparently not so rare in Italy.

It begins, as a rule, in early life in either sex, and shows a distinct hereditary tendency. Gilchrist found eleven cases in four generations in one family, and in other cases heredity has been traced.

Bacteriological examinations have been negative, although Wende after ten inoculations on the same patient succeeded with his last inoculation in producing what was apparently one of the lesions. This result was in the patient who already had the disease. Many inoculations in three other individuals were negative.

The process is a hyperplasia of the epidermis with hypertrophy of the mucous, granular, and horny layers. Wende found the increase of the horny layer so constant and marked—five to eight rows of cells—that he regards it as possibly the seat of the disease. The changes in the sweat glands are marked, and their openings are often filled by horny plugs. In the center of the lesions the sebaceous glands and the hair follicles are more or less atrophic. The papillary layer shows slight inflammatory changes, and in the central area may be obliterated.

Diagnosis.—The disease is unlike any other known dermatosis.

Prognosis and Treatment.—Upon the palms and soles the lesions from pressure and friction may become painful, but except for these secondary effects the disease gives no trouble. Although rarely lesions have disappeared (Joseph) they are usually persistent.

Excision of the lesions has proved successful, and Gilchrist found excellent results from the use of electrolysis. Curetting (Gilchrist) and the application of salicylic acid and other keratolytic agents (Joseph) have not proved successful in permanently removing the patches.

IX.—KERATOSIS PILARIS[1]

Synonyms: *Pityriasis pilaris, Lichen pilaris, Keratosis suprafollicularis.*

The term keratosis pilaris is applied to the roughening of the skin not uncommonly seen upon the extensor surfaces of the thighs and arms from the presence of horny papules formed by the accumulation of horn cells into plugs at the mouths of the hair follicles.

Symptomatology.—The condition is found to a greater or less extent upon the thighs and arms in almost all adults, especially during winter, when the cutaneous secretions are scant.

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[1] Brocq, *Annales*, 1890, pp. 25, 97, and 222 (review and bibliography).

The papules are acuminate or flattened, dry, rough to the feel, and rayish or, from the admixture of dirt at their tips, blackish. Occasionlly there is hyperæmia around their bases. The papules are usually enetrated by lanugo hairs which may be broken off. At times the plug revents the exit of the hair, and it curls up in the follicle and eventually roduces an inflammatory lesion which is familiar as ingrowing hair. he horny papule which is embedded in the follicle can with little difculty be picked off by the nail, leaving a conical depression. The deees of lichen pilaris vary greatly in different individuals, depending rgely upon the texture of the skin. Hardly any individuals except unger children are entirely free from some of the papules of lichen laris during the winter months. In dry, harsh skins the condition is ually abundant and in ichthyosis it becomes extreme.

Etiology and Pathology.—Its existence depends upon the texture the skin, and it is not a pathological process. Persons who sweat little more prone to it, and it is more marked in winter. It is apt to be re abundant in those who do not bathe frequently.

Anatomically the papule consists of an accumulation of horny epirmis around the pilo-sebaceous follicle. There is usually hyperæmia und the papules, probably as result of the mechanical irritation.

Diagnosis.—The condition is so familiar that it can hardly under linary conditions be confused with any other. Cutis anserina (goose sh), which produces an almost identical picture, is evanescent, while hen pilaris is permanent. When the papules have a congested or ghtly inflammatory areola they may be confused with the small folular syphilides or with lichen scrofulosorum, and possibly with pityisis rubra pilaris, but a brief reference to the characteristics of these eral affections is sufficient to differentiate them. The striking locan of keratosis pilaris especially upon the extensor surfaces of the arms d thighs alone distinguishes it from these other affections.

Treatment.—The horny plugs can readily be removed by frequent thing in warm water, especially when accompanied by scrubbing with een soap and a rough crash washcloth. Anointing the skin with a nd oil or fat in part prevents the formation of the papules, and is a eful measure in connection with bathing.

X.—LICHEN SPINULOSUS[1] (Devergie)

SYNONYM: *Lichen pilaris seu spinulosus* (Crocker).

Characteristics.—Under the second name given above Crocker deribes a rare affection consisting of an abundant eruption of follicular inflammatory pinhead-sized papules which are capped by horny spines. The papules are abundant and are collected in closely set groups and give the surface a nutmeg-grater feel. After a longer or shorter time the redness of the base disappears and the papules assume the normal color of the skin. The lesions may occur upon any part except the face, hands, and feet. They are usually most abundant over the extensor surfaces, but may be very abundant over the entire trunk. There is little or no itching, but some inconvenience from the very distinct spiny prominences.

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[1] Crocker, p. 452; *Atlas*, Plate XXXIV.—Adamson, *Brit. Jour. Derm.*, February, 05.

The description of the eruption does not clearly differentiate it from keratosis pilaris, in which the lesions not infrequently have minute inflammatory papules as their bases. The lesions in keratosis pilaris are also usually, while acuminate, not of the markedly spiny character described above.

Histologically the lesions are inflammatory infiltrations around the follicles with hyperkeratosis of the epidermal covering.

Prognosis and Treatment.—The lesions are apt to persist indefinitely if left to themselves, but readily yield to keratolytic applications. Repeated alkaline baths or thorough scrubbing with green soap to remove the horny plugs result in cure.

XI.—ICHTHYOSIS[1]

SYNONYMS: *Fishskin Disease, Xeroderma, Xeroderma ichthyoides, Ichthyosis vera.*

Ichthyosis (Greek, ιχθύς, a fish) is a congenital defect of the skin characterized by increase of the horny layer and deficiency of the cutaneous secretions.

It is not a very rare affection.

Symptomatology.—Ichthyosis, or ichthyosis simplex, is a general affection which involves the entire integument to a greater or less extent. Ichthyosis hystrix is an ichthyosis, limited to irregular circumscribed areas. Ichthyosis simplex is always meant when the unqualified term ichthyosis is used.

Ichthyosis varies in intensity from cases showing a slight abnormal roughening of the skin up to cases in which the horny epidermis consists of thick plates resembling the scales of amphibians. Ichthyosis is manifested most intensely upon the parts where the epidermis is normally roughest—that is, over the extensor surfaces of the elbows and knees and of the thighs and arms. It is less marked upon the front and back of he trunk, the face and the scalp, and, except in the severest cases, the rger flexures are slightly if at all involved. But even on the parts where he skin is apparently not involved in ichthyosis it is usually drier and arsher than normal. It is not known to affect the mucous membranes xcept in a single case reported by Thibierge,[1] in which there was inolvement of the mucous membranes of the mouth and nose. In all ases of ichthyosis keratosis pilaris is usually prominent. Ichthyosis, hile a congenital efect of the skin, oes not ordinarily ecome sufficiently eveloped to be aparent until the secnd or third year. fter reaching a cerain stage in early hildhood it usually ersists with only light changes durng life. At puberty, rith the normal inrease in the activty of the sebaceous lands which occurs hen, there may be ermanent improvenent in ichthyosis, ut on the other hand he slightly harsher pidermis which haracterizes adult ife may counteract his favorable influnce and render the ondition worse than luring childhood. The condition, as a rule, improves during warm veather when the skin is active and the air moist, and becomes worse luring the winter when the air is dry and the skin less active.

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[1] Fox, G. H., *Jour. Cut. Dis.*, 1884, p. 97.—Lebert, "Ueber Keratose," Breslau, 1864.

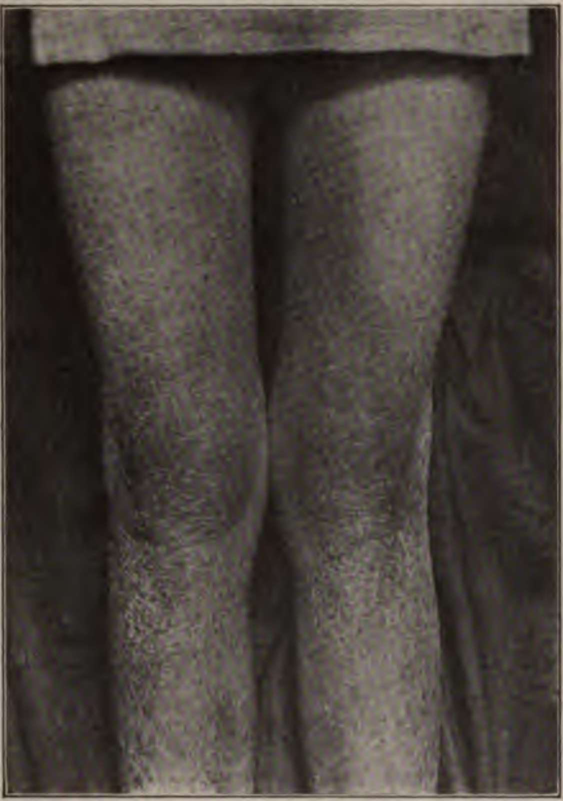

FIG. 237.—XERODERMA. (Author's collection.)

The hair and nails usually participate to some extent with the horny pidermis. The hair is dry and lusterless, and the nails to a greater or ess extent are friable, opaque, and stunted.

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[1] *Annales,* 1892, p. 717.

Jadassohn,[1] Hallopeau and Jeanselme,[2] and Audry[3] have described cases of ichthyosis in which the skin as a whole was atrophic.

Ichthyosis itself is without subjective or constitutional symptoms. Even in the mildest cases, however, the skin from defective secretions is more vulnerable than normal, and ichthyotic patients therefore are usually troubled more or less with dermatitis, especially in cold weather. In the extreme cases, on account of the deficiency of mobility of the skin, painful fissures are common.

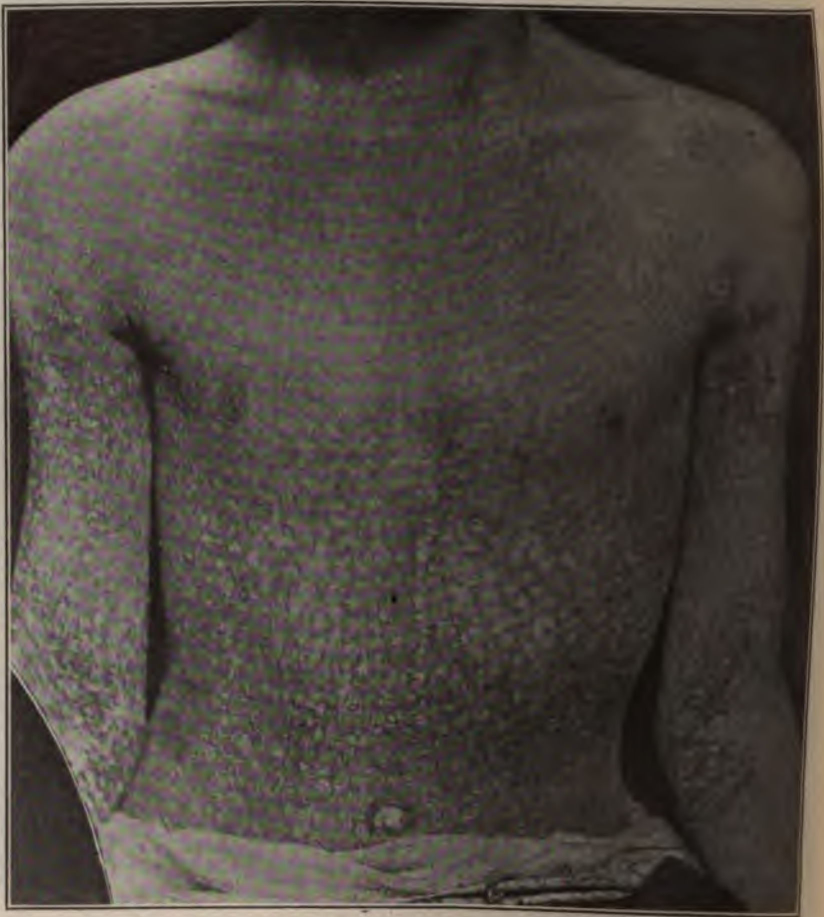

FIG. 238.—ICHTHYOSIS OF MODERATE DEGREE. (Author's collection.)

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[1] *Verhandl. der IV. Deutsch. Dermatolog. Gesellsch.*, 1894, p. 392.

[2] *Annales*, 1895, p. 1016.

[3] *Jour. mal. cutan.*, 1895, p. 265.

Xeroderma.—The slightest degrees of ichthyosis are called xeroderma. In these cases the skin as a whole is harsher and rougher than normal, with distinct thickening of the horny epidermis over the elbows and knees and on the extensor surfaces of the thighs and arms. Over these reas of greatest intensity there is some free exfoliation of horny scales, nd from the exaggeration of the normal markings the surfaces have a essellated appearance. Over the elbows and knees the scaling is thrown p into rough hyperkeratoses, perhaps in transverse ridges, and is blackh and dirty looking.

Ichthyosis Simplex.—In the more marked cases, to which the term hthyosis is commonly confined, the horny epidermis is a thick harsh yer which fractures in various directions, and by these superficial cracks so marked that it has a resemblance to the scales of a fish, from which he affection gets its name. The large scales curl up at their edges in hese epidermal cracks, while toward the center the scales are adherent. he exfoliation of epidermis is abundant, and the scales rub off freely pon the clothing. The skin is more markedly gray and dirty looking, nd over the elbows and knees the horny epidermis is thrown up into idges of hyperkeratosis of dark-gray or black color. In many of these ases the flexures escape, but the skin of the face and hands is apt to how appreciable evidence of the condition, and is often, especially in old weather, eczematous.

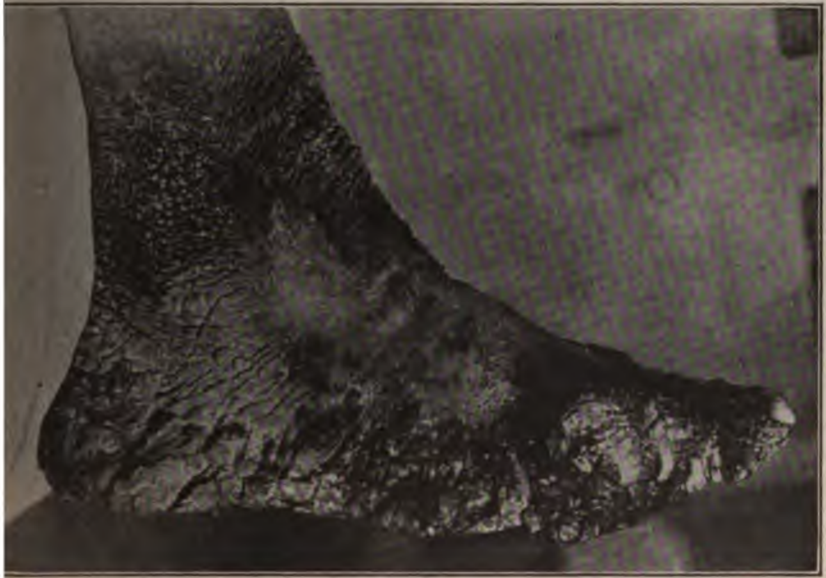

FIG. 239.—ICHTHYOSIS, EXTREME DEGREE. (Engman and Mook's collection.)

In extreme cases of ichthyosis the epidermis consists of thick horny plates divided into polygonal figures by coarse markings. The plates are gray to blackish, and the involved skin is like that of a thick-scaled animal. In these cases the parts where the skin is normally thinnest present the appearance of a slighter degree of ichthyosis. To these extreme cases are sometimes applied various fanciful names: *ichthyosis serpentina,* where the resemblance is supposed to be to the scales of a serpent; *ichthyosis sauroderma,* or *sauriasis* (crocodile skin); *ichthyosis scutulate,* where the lesions are shield shape.

Ichthyosis Congenita[1] (*Keratoma diffusum; Intrauterine ichthyosis; Harlequin fœtus*).—In rare cases ichthyosis is manifested at birth. Such cases are ordinarily extreme examples of the condition, with the surface covered by thick, scalelike plates, marked in all directions by fissures in the horny epidermis. There is more or less distortion from the immobility of the skin; there may be ectropion, immobility of the mouth, and fissures at all of the external orifices. Such children are frequently prematurely born, and usually die within a few days or weeks from inanition or loss of heat. A cause contributory to their death is not infrequently the impossibility of nursing on account of the immo-

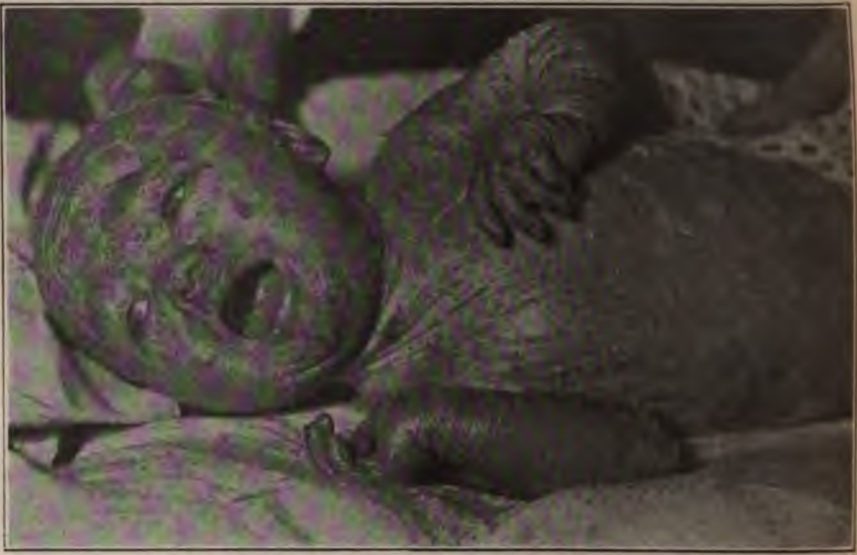

FIG. 240.—ICHTHYOSIS CONGENITA. (Grindon's collection.)

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[1] Meneau, *Annales,* 1903, p. 97 (complete bibliography and review).—Caspary, *Archiv,* vol. xiii, 1886 (bibliography).—Elliot, *Jour. Cut. Dis.,* 1891.—Sherwell, *ibid.,* 1894, p. 385.—Winfield, *ibid.,* 1897, p. 516.

lity of the lips. These cases of congenital ichthyosis are true ichthyosis, e most exaggerated manifestation of the condition.

There are also other cases which simulate but are not true ichthy-sis: Hebra and Kaposi's ichthyosis,[1] *ichthyosis sebacea, or seborrhœa quamosa neonatorum*, is essentially an abnormal accumulation of vernix aseosa, which continues to be produced for a few days after birth. The kin is encrusted and tense, with painful fissures, and of a brownish-red olor which looks like a half-browned sucking pig (Hebra). The chil-ren die from inanition unless the encrustations are softened and re-oved by inunctions and the body temperature artificially preserved.

In other cases the child is born with a membrane covering the skin hich resembles collodion or oiled paper. Soon this peels off in sheets, ter which there is moderate desquamation which disappears leaving the rmal skin. Such cases have been recorded by Hallopeau,[2] Grass and örök,[3] and Bowen.[4] Bowen believes they are "examples of a persistence the epitrichial layer, which has usually been cast off by the seventh tal month, but in these instances maintained its integrity up to the me of birth."

Stelwagon suggests that some of the other cases of congenital ichthy-is in the literature are probably examples of exfoliative dermatitis.

Ichthyosis hystrix is applied to cases in which there exist circum-ribed patches of extreme ichthyosis. The condition is quite analogous nævus. It indeed may well be regarded as horny nævus, just as gen-alised ichthyosis is a congenital defect which may be compared to a iversal horny nævus. The patches of ichthyosis hystrix show all de-ees of horny hypertrophy. They are roughened, papillated, and cov-ed by thick horny masses which at times may be thrown up into sharp ines. The patches are more or less elevated and wartlike, reaching it ay be half an inch or more above the surrounding skin. In shape and ze they are altogether irregular, as they are in distribution. There ay be one or several affected areas. Between the patches the skin is dinarily of normal texture, but—and this shows the essential identity the affection with simple ichthyosis—in some of the cases the re-ainder of the skin has shown the changes of ichthyosis of less intensity.

Ichthyosis hystrix linearis, or linear ichthyosis, differs clinically om ichthyosis hystrix in that it occurs in bands or streaks which fol-w more or less closely the course of cutaneous nerves.

These cases have been described under various names—*linear nævus,*

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[1] Kaposi, p. 125.—Sutton, *London Med.-Chirurg. Trans.*, 1886, p. 291 (bibli-raphy).

[2] Hallopeau and Watelet, *Annales*, 1895, p. 149.

[3] *Annales*, 1895, p. 104.

[4] *Jour. Cut. Dis.*, 1895, p. 485.

nævus unius lateris, nævus nervosus, nævus lichenoïde, ichthyosis linearis neuropathica, papilloma lineare, papilloma neuropathicum unilaterale, nævus verrucosus, nævus papillaris, papilloma neuroticum, etc. Whether they are regarded as a form of verrucose nævus or of ichthyosis hystrix is a matter of names. Their identity with the lesions of ichthyosis hystrix is shown by the exact correspondence of the individual lesions in both affections.

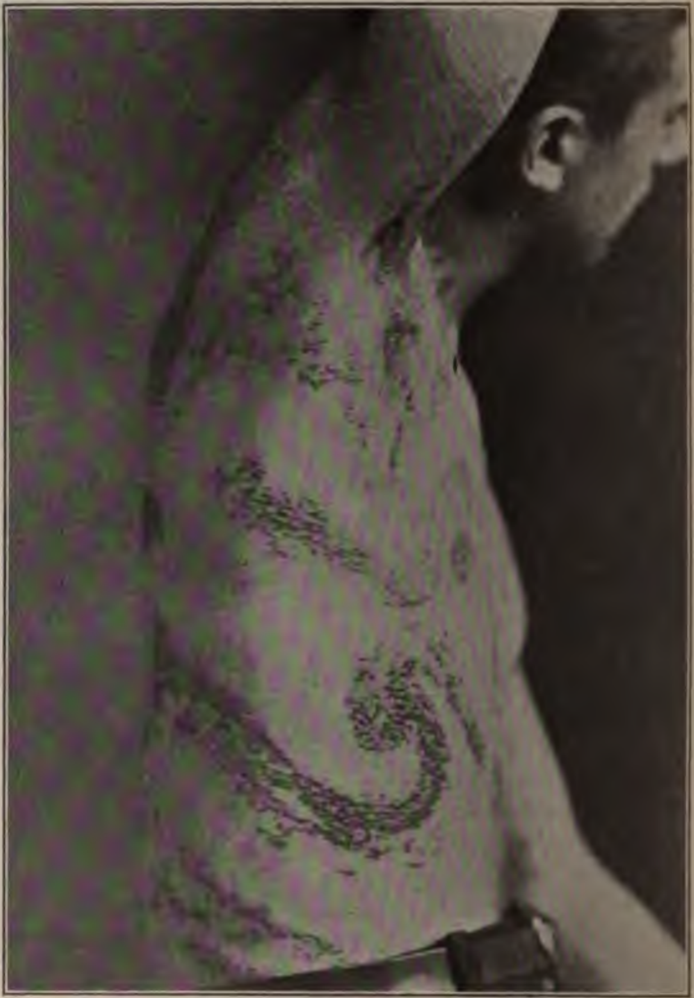

FIG. 241.—NÆVUS LINEARIS. (Winfield's collection.)

Linear ichthyosis is usually unilateral, although not always so. It appears in bands, following the axis of the limbs or the lines of cleavage ound the trunk. The degree of its manifestations varies as it does in hthyosis hystrix.

There has been much discussion as to the reason for the peculiar stribution of the lesions of linear ichthyosis, as there has been for ost other cutaneous lesions which follow more or less closely the course cutaneous nerves. These lesions do not exactly follow the course cutaneous nerves, so their distribution is not to be explained upon rve influence alone. Other explanations are that they follow the es of cleavage of the skin, or the course of blood vessels, or the etameres or segments of the body, or that they lie along the nbryonic sutures and follow the trend of growth of the tissues. . W. Montgomery,[1] who has carefully considered the subject, re- rds the last theory as the one "that most nearly fulfills all the quirements."

Acquired Ichthyosis.—A condition clinically the same as ichthyosis s been observed by Crocker to develop in one patient at thirty-six, in second at sixty-four, and in a third at seventy-six years. Similar cases ave been reported by Tommasoli and by Mapother, and acquired ich- yosis is said by Crocker to be common in natives of the Sandwich lands who chew piper methysticum. The essential identity of these ses with true ichythyosis is open to question.

Circumscribed areas of ichthyosis may develop in connection with euritis and in tabes. These, as Unna suggests, may be classed with e hyperkeratoses which are seen with varicose ulcers, chronic eczema, nd elephantiasis, and differ distinctly from true ichthyosis in their rigin in chronic inflammatory processes.

Chronic palmar and plantar ichthyosis is keratosis palmaris et lantaris.

Ichthyosis linguæ is leukoplakia.

Etiology and Pathology.—Eliminating the acquired cases of oubtful character, ichthyosis is always congenital, but as to the reason or its occurrence we know nothing. Although exceptions occur, the ffection, as a rule, is markedly hereditary. The heredity may be direct r collateral. Occasionally several members of a family will show the ondition; in other cases only one will be affected. There is no predi- ction for either sex, but in family groups of cases there may be a dis- nct tendency to affect one sex to the exclusion of the other. Kaposi as recorded a group of cases in which an ichthyotic mother had eight ildren: five sons who were ichthyotic, and three daughters who were ree.

The pathological anatomy of ichthyosis has been studied by Esoff,[2]

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[1] *Jour. Cut. Dis.*, 1901, vol. xix, p. 455.

[2] Virchow's *Archiv*, 1877, p. 417.

Audry,[1] Tommasoli, Unna,[2] and others. Changes in the corium are not marked. There may be a slight chronic inflammatory infiltration in the papillary layer and around the follicles, consisting of leucocytes and round cells; plasma cells are absent and mast cells very few. The papillæ are flattened and pressed apart by the pressure of the epidermis, the processes of which fit in between them, producing the appearance of roughly rectangular projections dovetailed into each other. The collagen in the papillary layer is increased. The principal change is in the epidermis, and consists of an increase in the horny layer which may be as great as two hundred times. The horny layer is formed in a peculiar way directly from the prickle-cell layer without any preliminary appearance of keratohyalin or eleidin granules, the granular layer being absent. The prickle-cell layer is thinner than normal by reason both of this rapid change into horny cells and also of atrophy due to the pressure of the overlying corneous layer. The glands are unaffected except for dilatation of their lumina.

According to Kaposi the histological structure of ichthyosis hystrix is entirely similar to that of verruca. Crocker's investigations showed in addition a dipping down of the corneous layer into the interpapillary spaces, following the outline of the papillary layer.

Diagnosis.—The clinical features of ichthyosis are distinctive, and there is no other condition which closely resembles it. Xeroderma might be confused with prurigo and pityriasis rubra pilaris, but a review of the characteristics of these affections would render the distinction easy. The question of the distinction between ichthyosis hystrix and warty nævus and between ichthyosis linearis and nævus unius lateris might come up, but, as has been indicated, is a matter of names. The warty hypertrophies which occur at times in chronic eczema of the legs and in elephantiasis are distinguished by the fact that they are acquired and are the result of chronic inflammatory processes.

Prognosis.—Ichthyosis may be improved, but is a permanent deformity of the skin which cannot be cured. The only recorded cases in which recovery has taken place are two by Hebra,[3] one, a child of eight in which the condition disappeared permanently after an attack of measles, the other a case of ichthyosis hystrix which disappeared after an attack of variola.

Treatment.—Internal treatment is only palliative while it is continued or its effects persist, and it is therefore useless. From stimulation of the sweat secretion by pilocarpine there is usually some temporary improvement. More benefit can be obtained by the use of thyroid extract, as shown by the reports of Bramwell,[1] Abraham,[2] and others. When thyroid extract is used it should be given in small doses, beginning with 1 or 2 grains daily for a child and 5 grains for an adult, to be increased cautiously to twice these doses.

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[1] *Annales*, 1893, p. 354.—*Annales*, 1893, p. 537 (bibliography).

[2] "Histopathology," p. 322.

[3] Hebra and Kaposi, "Hautkrankheiten," vol. ii, 1876, p. 41.

The local treatment consists in measures to get rid of the excess of horny epidermis and to keep the skin lubricated and pliable. These measures have to be continued indefinitely, but after the condition of the skin has been improved by vigorous treatment, the improvement can be maintained by much less constant attention. In mild cases it is sufficient for the patient to have daily baths followed by anointing with a bland fat. Simple warm alkaline soap or bran baths may be sufficient, but when a stronger effect is desired the patient should first cover his body thoroughly with green soap, after which he takes a bath for ten or fifteen minutes in warm water with vigorous scrubbing. After the epidermis is softened by the bath the skin should be thoroughly greased with a neutral fat. For this purpose any of the ordinary oils or fats are satisfactory—olive oil, benzoinated lard, cold cream, or mixtures of vaseline or olive oil (2 parts) with lanolin (1 part). For the face and hands the same applications may be used. An agreeable application for these parts is an emulsion of lanolin in limewater or rose water in the proportion of 1 : 10. Glycerine and sodium biborate, each ten per cent in water, is also an agreeable application for the hands and face. By assiduous attention to bathing and greasing the skin it may be apparently restored to normal in mild cases and kept in this condition by further attention.

In the severer cases, in addition to the bathing, preferably with green soap as described above, it may be necessary upon the parts where the hyperkeratosis is greatest to use keratolytic salves; and for this purpose salicylic acid, as usual, is the most satisfactory agent. It may be used in the proportion of two to ten per cent in any of the ordinary ointments. The combination of resorcin and salicylic acid, each two to ten per cent, is also highly recommended.

In ichthyosis hystrix radical measures are necessary to get rid of the lesions. The lesions may be macerated by prolonged scrubbing with green soap and subsequent bathing in warm water, after which a salicylic-acid ointment of ten- to twenty-five-per-cent strength may be applied to the surface, and this treatment continued from day to day until the horny masses are dissolved away. The horny masses may also be dissolved away by repeated washing with liquor potassæ. After the horny covering is removed, if permanent results are to be obtained, the hypertrophied papillæ have to be destroyed and scar tissue formed to take their place. The destruction of the papillæ may be caused by the application, as suggested by Crocker, of a saturated solution of salicylic acid in alcohol, or by cauterization superficially with chloride of zinc, or by the actual cautery, or by vigorous curetting. By tentatively carrying out such vigorous methods of treatment great improvement may be produced in ichthyosis hystrix, but it involves the production of scar tissue in place of the congenital lesion, and must therefore be carried out cautiously. When the lesions are not a source of great disfigurement or inconvenience they are better left alone.

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[1] *Brit. Jour. Derm.*, 1894, p. 205.

[2] *Ibid.*, 1896, p. 106.

XII.—NÆVUS[1]

SYNONYM: *Birthmark.*

Nævi (Latin, *nævus*, a mask) are of two kinds—pigmented and vascular. They are both anomalies of development of the skin, one showing chiefly in excessive deposit of pigment, the other in overgrowth of vascular tissue. Vascular nævus is taken up along with other vascular overgrowths of the skin. Pigmented nævus is taken up here in connection with ichthyosis hystrix, with which it presents many features in common.

XIII.—NÆVUS PIGMENTOSUS

SYNONYM: *Pigmented Mole.*

Pigmented nævi are congenital circumscribed lesions of the skin in which there is increased deposit of pigment, either with or without increase in the other elements of the skin. The lesions are usually associated with hyperplasia of the derma as well as of the epidermis and frequently with overgrowth of hair.

Symptomatology.—Pigmented nævi are moles. Ordinary small moles are familiar lesions. The largest nævi are identical in character with these smaller lesions, but differ in exaggeration of their features.

The simplest form of pigmented nævus is the flat mole, a circumscribed deposit of pigment in the skin of light- to dark-brown color. Such lesions may be without elevation—*nævus spilus* (from Greek σπίλος, a spot)—or slightly elevated. As a rule they are small, from an eighth to a half an inch in diameter, but occasionally they may be as large as a hand or even much larger. In the next type of nævus there is some e of the derma and the lesion is elevated. It may be quite smooth urface may be pitted somewhat by large follicles, or mammillated ypertrophy of the papillæ. In any case there may or may not be ıormal growth of hair.

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[1] The number of cases of nævus of extensive areas or of peculiar distribution or presenting some other peculiar features which exist in the literature are almost unlimited. The histology of the lesions and of malignant growths arising from them have been studied especially by Gilchrist, *Trans. Amer. Derm. Assn.* for 1898; Whitfield, *Brit. Jour. Derm.*, 1900, p. 268 (bibliography); Sachs, *Archiv*, 1903, Bd. lxvi; Johnston, *Jour. Cut. Dis.*, January and February, 1905 (complete bibliography); W. S. Fox, *Brit. Jour. Derm.*, January and February, 1906.

er cases there is some e of the horny layer as hypertrophy of the pa- ınd the lesions are wart- *ıævus verrucosus*). At he papillary hypertrophy ıarked feature and pro- the type of lesions de- as *nævus papillomatosus.* papillomatous and horny ıay have a very offensive om the decomposition of nd cutaneous secretions. nævi, except those with- vation, show an abnor- owth of hair, perhaps ıt but usually of dark This growth may be ıt to make the nævus as ly a hairy lesion as a ted one (*nævus pilosus*). nally the lesions show a increase in the amount ıneous fat, so that ex- r their increased pigmentation they resemble lipomatous tumors *lipomatodes*).

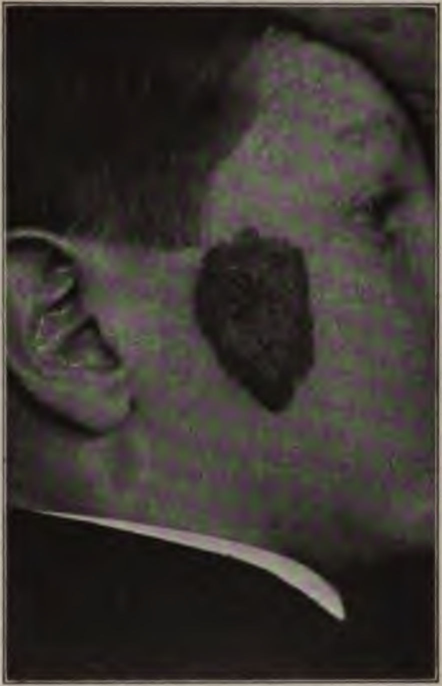


FIG. 242.—NÆVUS PIGMENTOSUS. (Author's collection.)

size, shape, number, and distribution of nævi there is the widest ɒn. Nævi rarely exceed the size of a hand, but in occasional cases ıvolve extensive areas. Linear nævus has been considered under ısis hystrix linearis. The other features of pigmented nævi usu- ry in the same ratio with the size, so that in the larger nævi we eater elevation, a greater amount of hyperkeratosis, a more papil- us or warty surface, and a more exaggerated growth of hair. The r of nævi is unlimited. There may be only one or two, and in ıstances the large nævi are fortunately limited to one or a few iven case. Frequently, however, where there is no large nævus re many moles over the body, and in other cases large nævi may ciated with many moles or several large lesions may exist in the ase.

Nævi occur upon any part, but their sites of predilection are the face, especially the forehead, the neck, and the back. Their distribution is irregular, but they are usually distinctly asymmetrical. Their distribution often suggests the influence of cutaneous nerves, or of blood vessels, or of the metemeric segments of the skin, or of embryonic sutures

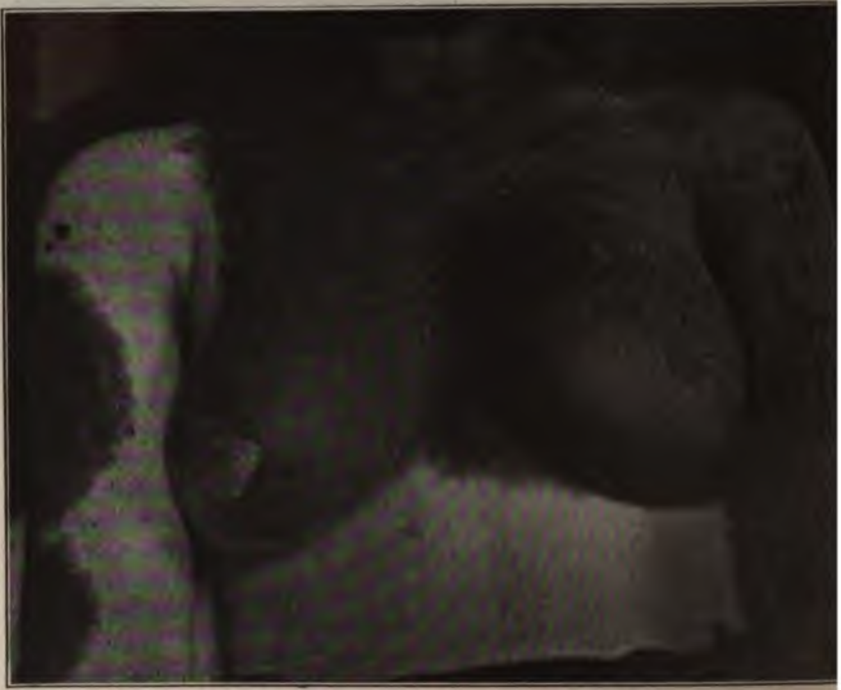
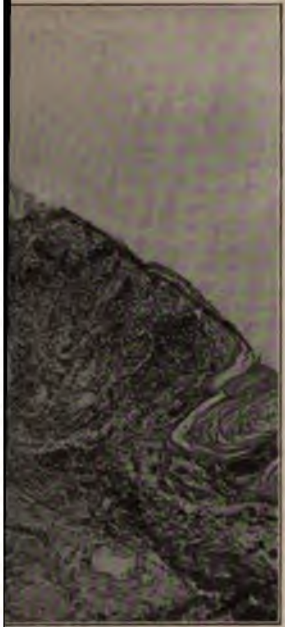


Fig. 243.—Nævus Pigmentosus Pilosus. (Heidingsfeld's collection.)

These relations, however, do not usually bear close analysis, and wha the relation is, if any, is not established. Upon this point what ha been said of the distribution of ichthyosis hystrix applies equally well t pigmented nævus.

Moles, like ichthyosis, although congenital may not be apparen until months or a year or two after birth. They most frequently appea in the course of several weeks after birth. Moles sometimes apparentl develop long after birth. In these cases the probable explanation is tha the embryonic defect existed from the start, but only gradually becam sufficiently marked to be apparent. After their full development the do not, as a rule, increase in size. This, however, may happen. Thei other characteristics usually become more prominent with the growt of the patient, so that a nævus at first smooth may later become papillary, rough, and horny, and show a coarser growth of hair. They las

PLATE IV



ÆVUS PIGMENTOSUS. Dark
nelanin. × 65. (Author's
)

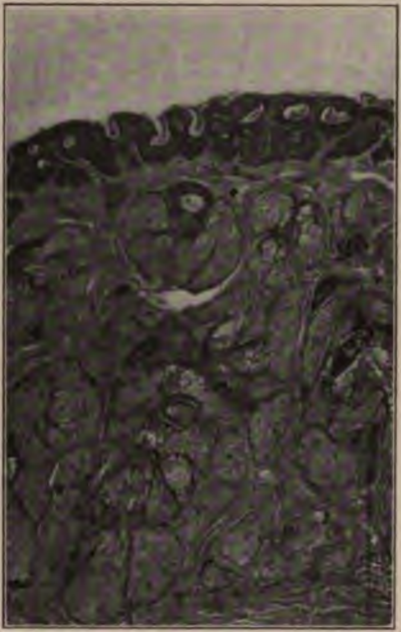
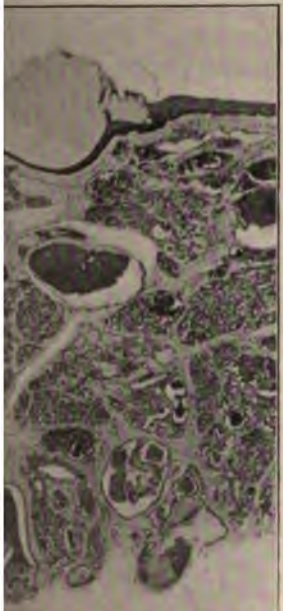


FIG. 2.—NÆVUS SEBACEUS. × 30.
(Author's collection.)



LE. Anatomically a hæman-
dematous from constriction;
œdema on surface. × 30.
collection.)

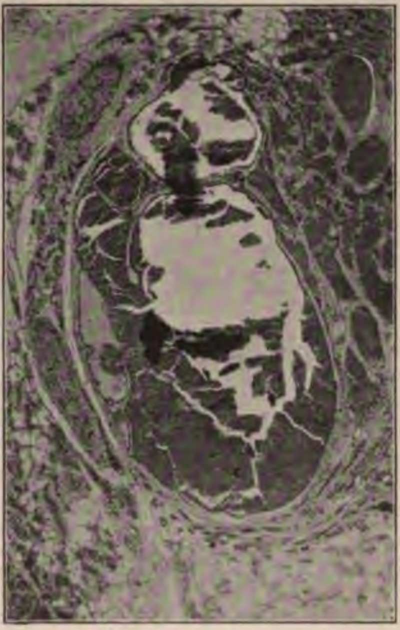
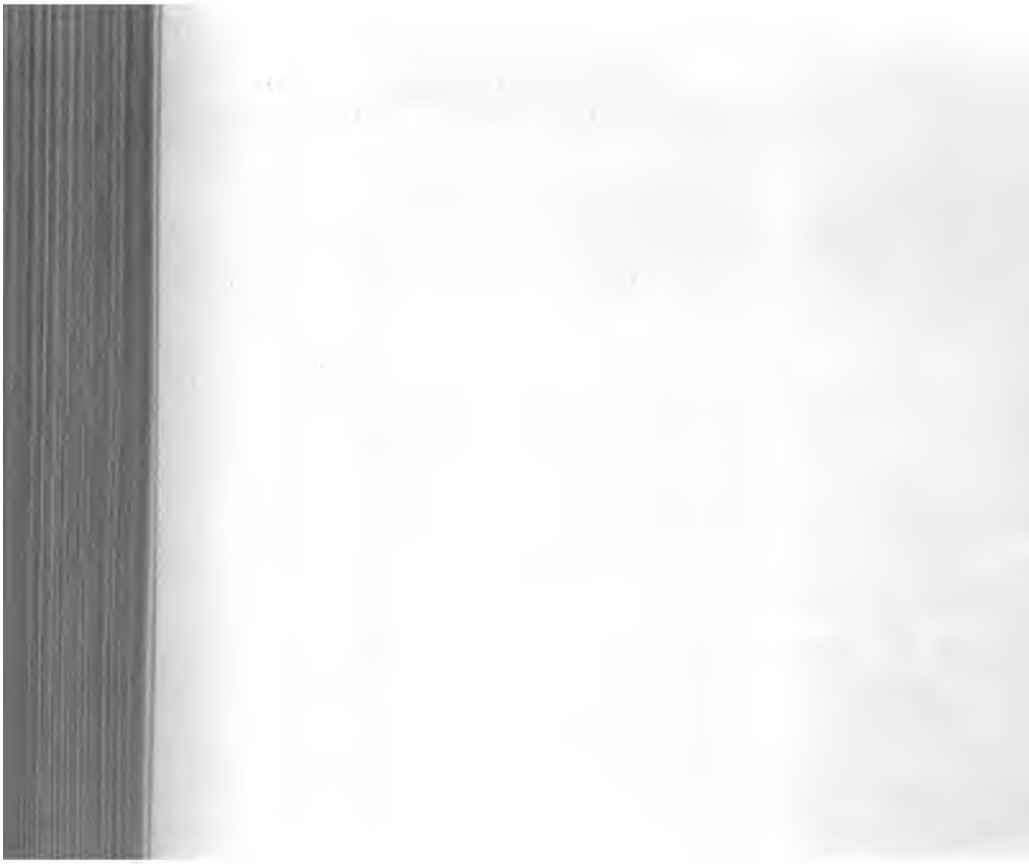


FIG. 4.—CAVITY IN A VASCULAR NÆVUS.
× 40. (Author's collection.)

MICROPHOTOGRAPHS OF NÆVI



hrough life, and do not tend to disappear spontaneously, although pitzer[1] has reported the spontaneous involution of a giant nævus.

There is a more or less dangerous tendency for moles, especially if rritated, to become malignant. This tendency increases with years, ut may show itself early, and when they become malignant they prouce melanotic growths of extreme malignancy.

Histology.—The microscopical anatomy of moles and other nævi is ighly variable. The lesion may consist of hypertrophy of almost any of ie tissues of the skin. In vascular nævi it consists chiefly of hyperophy of the vascular tissue. In pigmentary and horny nævi there is n enormous increase of the horny layer of the epidermis and of the pigent in the epidermis and in the corium. In the soft white moles it nsists of a great increase of endothelial cells which are arranged in lumns and strings, as shown in the illustration given under endoelioma. In other cases the hypertrophy may consist largely of an normous increase in the size and number of the hairs, along with ypertrophy of the pigment and of the horny layer. In still other cases ie hypertrophy consists almost entirely of an increase in the sebaceous lands. In reference to the soft moles which I have described as made f masses of endothelium it should be said that they are regarded by nna, Hutchinson, Jr., Gilchrist, and others, not as endothelial strucures, but as epithelial. Virchow, Kaposi, and the majority of patholoists regard them as endothelial tumors.

In the pigmented nævi melanin is present in greatly increased mount in the deeper epidermis, and the fibrillated structure of the rickle cells is lost to a great extent; to this and the resultant "state of nstability" MacLeod attributes the tendency of these moles to become alignant.

Treatment.—Moles if showing evidences of irritability or if exosed to frequent external irritation should be removed. From the langer of malignant transformation in them, if they are treated, the reatment should be radical enough to cause their immediate destrucion, and for this purpose clean excision forms always the best method. They may also be destroyed with the strong caustics. A convenient nethod of destroying small nævi is electrolysis. When this is used it hould be used thoroughly enough to completely destroy the lesion or he part of it which is treated. One or two unfortunate cases within my nowledge, in which melanomas have developed in pigmented moles reated by electrolysis, have made me regard it as less satisfactory than nore vigorous methods of treatment.

The treatment of large pigmented nævi, especially when they are hypertrophied and hairy, as they usually are, is a difficult problem. They may be treated by electrolysis, removing the hairs in the usual way and also destroying the hypertrophied tissue by longitudinal insertions of the needle, but this method is so interminable that it is practically ineffective. The best results which I have seen in such nævi have been in a few cases from the use of x-rays alone, and in others from operation followed by x-rays. In some cases I have gotten from x-rays excellent results—complete disappearance of the hair and almost complete disappearance of the pigment; but in coin-sized nævi this method, in my experience, has been followed by depressed scars which are somewhat disfiguring. The excision of a large nævus with a plastic operation or with simple skin grafting usually results in hypertrophic, disfiguring scars; but in several cases of this sort I have been able to cause thinning of the scars with x-rays and to substitute a smooth, white, pliable, uncontracted scar which has made the results satisfactory. In the last three years I have treated many pigmented nævi with CO_2 snow, by freezing from ten to sixty seconds. Small nævi can be covered with one application. In large nævi it is better to freeze adjacent areas by successive applications. One freezing may be sufficient, but it is better not to overdo the freezing and to repeat the applications, if necessary. The same results can be obtained with liquid air. These two methods give us fairly effective means of treating large pigmented nævi. Results are obtained that are better than those from any other method of treatment.

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[1] *Derm. Zeitschr.*, January, 1905.

XIV.—KERATOSIS FOLLICULARIS[1] (White)

SYNONYMS: *Keratosis vegetans* (Crocker), *Ichthyosis follicularis*, *Darier's Disease*, *Psorospermosis*, *Psorospermose folliculaire végétante*, *Acné sébacée cornée*, *Ichthyoṣis sebacea cornea* (E. Wilson).

Keratosis follicularis is a disease characterized by a peculiar form of hyperkeratosis beginning in, but spreading beyond, the openings of the pilo-sebaceous follicles, and producing peculiar symmetrically distributed conical and papillomatous horny lesions.

The disease was first described in 1889 by J. C. White and Darier independently.

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[1] Darier and Thibault, *Annales*, July, 1889.—White, J. C., *Jour. Cut. Dis.*, June, 1889, and January, 1890.—Bowen, *ibid.*, 1896, p. 209 (review and bibliography); *Annales*, 1898.—Hallopeau, *Annales*, 1896.—Omerod and McLeod, *Brit. Jour. Derm.*, 1904 (review and complete bibliography).—Bering, *Archiv*, 1905, vol. lxxiv. —Curl, *Jour. Cut. Dis.*, 1905, vol. xxiii.—Malinowski, *Monats.*, September, 1906 (review and bibliography).

Symptomatology.—The disease is essentially a hyperkeratosis ound the openings of the sebaceous and hair follicles, and in its first anifestations is indistinguishable from thickly studded lesions of kera-

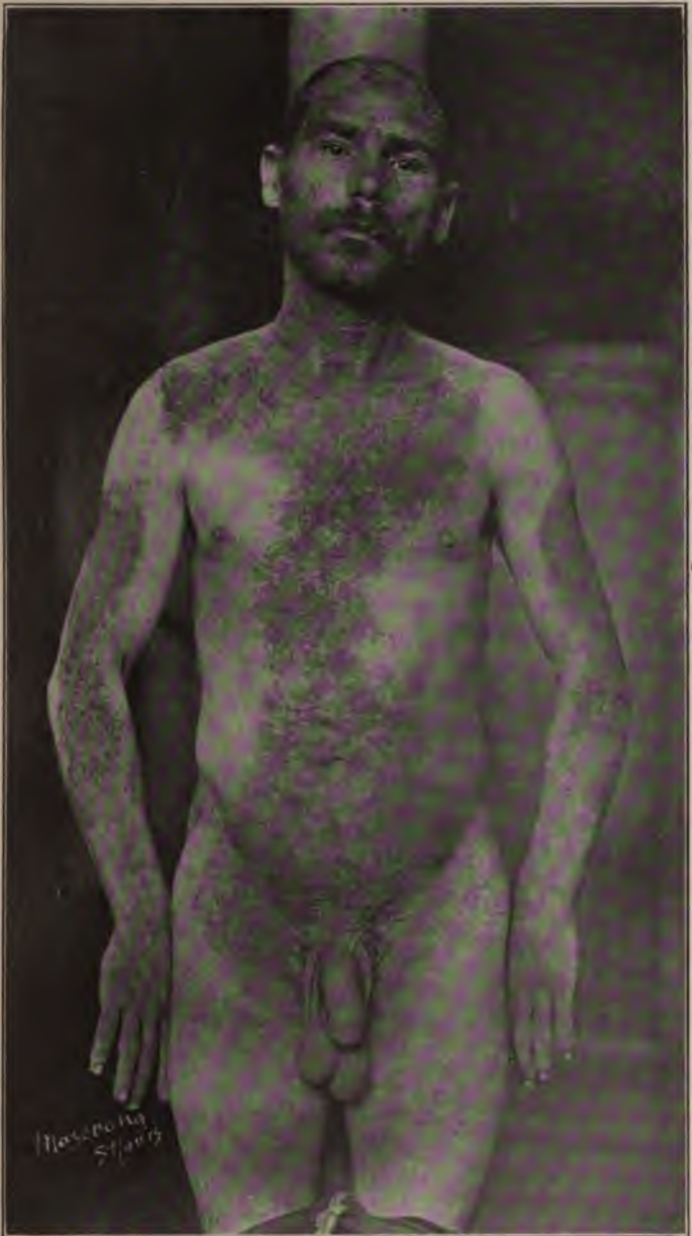

Fig. 244.—Keratosis Follicularis. (Grindon's collection.)

tosis pilaris. Later the lesions increase and become greasy looking, grayish or brownish, globular papules of pinhead size or larger. These lesions on examination with a hand lens show semicornified sebaceous concretions embedded in their apices. Later some of the lesions become globular or flattened large papules of dark-brown to dull-red color, and with or without the central concretions. Very advanced lesions are hard and horny, of dark gray or brown color and of the size of a pea. Where the lesions are abundant and become largest they coalesce into rough papillomatous masses covered by a thick grayish or brownish horny layer. Occasionally the horny hypertrophy is exaggerated, producing truncated conical lesions a quarter to a third of an inch in diameter and a quarter to a half an inch in height. In the earlier stage of development the lesions may, instead of showing as papillary elevations with central concretions, be crateriform follicular openings filled with the usual concretions. In these dilated follicular openings there may be infection with ulceration and purulent discharge. In a well-developed case lesions in all stages of their evolution will be seen.

The disease occurs as an abundant thickly set eruption with well-marked predilection for certain parts. These are in general parts where the development of sebaceous glands is abundant; the glabella and the temples near the border of the hair, the cheeks around the alæ nasi, the ears (including the external auditory canal), the trunk, especially the sternal, the interscapular, and the sacral regions and the median line of the abdomen, the vulva, and the arms and legs, the hands and feet usually escaping. Where the lesions are abundant and coalesce into hypertrophied papillomatous masses there is frequently an intolerable odor of decomposing horn and sebaceous matter. On the scalp the lesions frequently take the form of plugged dilated sebaceous follicular openings which may be suppurating; and the scalp as a whole is often covered by a layer of greasy scales resembling seborrhœic eczema.

The course of the disease is persistent and slowly progressive. In none of the cases has there been the development in the lesions of epitheliomata, a possibility that might be expected (Bowen).

There are, as a rule, no subjective symptoms. The general health is undisturbed.

Etiology and Pathology.—The affection is exceedingly rare, about twenty-four cases being recorded.

It ordinarily begins in childhood; indeed, Bowen suggests its close resemblance to the anomalies of development like ichthyosis, and its probable existence in most cases for a considerable time before attention is paid to it. About eighty per cent of the patients have been males. White, Boeck, and Ehrmann have had two or more cases in one family, but beyond these cases there is no evidence either of heredity or of contagion.

Darier found coccidialike bodies in the lesions which he thought were sorosperms, but later investigations of Piffard, Bowen, Darier himself, nd others have shown that these were atypical forms of cornified epihelial cells.

Histological examinations by Bowen, Darier, and Lustgarten have hown that the process is an epithelial hyperplasia beginning in the neck f the pilo-sebaceous follicles and resulting in a peculiar parakeratosis. n the late large lesions the process extends in the epidermis entirely eyond the follicular openings. In the epidermis and corium at the order of the lesions there is increased deposit of pigment. In the large morlike lesions there is abundant proliferation of the rete downard into the corium. In the corium there is only slight cellular filtration.

Diagnosis.—The diagnostic features of the disease are the combinaon of lesions like those of keratosis pilaris, larger papular hyperkeraoses around the follicular openings with central concretions, and, where he lesions are abundant, the formation of tumorlike rough papillomaous masses. In addition the eruption is abundant, symmetrically disributed in its areas of predilection, and persistent.

Prognosis and Treatment.—The disease is extremely rebellious to reatment, and nothing can be done for it except palliation. The conition can, however, be greatly improved by local treatment. This onsists in frequent alkaline baths, especially baths with green soap and he use of salicylic acid, sulphur, and resorcin ointments, as in the treatment of ichthyosis. Large horny masses are treated in the same way s the horny masses of ichthyosis hystrix.

Herxheimer[1] has recently recommended the thermocautery for the estruction of the lesions of keratosis follicularis. In this way he has eated three cases. Without the use of an anæsthetic, the keratoses ere cauterized individually. No dressings were applied. In three ttings, he was able to treat the entire diseased area, and in this way o of the patients have been cured. One of the cases has remained well veral years, the other several months.

XV.—KERATOSIS FOLLICULARIS CONTAGIOSA

Under this name Brooke[2] has described a disease which resembles ore or less closely keratosis follicularis, but is apparently contagious.

Symptomatology.—The lesions occur as an abundant crop of dry, orny plugs filling the sebaceous follicles. They may be thin, sharp spines deeply embedded in the follicular openings, or fleshy papules, with an inflammatory base resembling acne papules but filled with adherent, dry, horny, instead of greasy sebaceous plugs. The plugs are black and dirty looking, and the skin as a whole in the affected areas has a yellowish tinge, the combination giving the parts a dirty blackish color resembling somewhat ichthyosis. The eruption has a symmetrical distribution, and on the parts involved the lesions are very abundant, but they remain discrete and there is no confluence into the papillomatous masses seen in keratosis follicularis. The areas of predilection of the lesions are the back of the neck, where they usually first appear, the arms, forearms, and hands, and the posterior axillary fold. In the case illustrated by Brooke they spread to the trunk, the sides and front of the chest, the front of the neck, and the face. They also involved, but less markedly, the flexure surfaces of the legs, the inner sides of the arms, the buttocks, and the lower part of the back.

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[1] Herxheimer, *Derm. Zeitschr.*, 1908, p. 45; *Brit. Jour. Derm.*, 1908, p. 170.

[2] "International Atlas of Rare Skin Diseases," Part VII, Plate XX.

The eruption in most cases after appearing upon the neck spreads in the course of a few weeks to other parts. The eruption is persistent without treatment, but readily yields to keratolytic applications, and there is a tendency to spontaneous recovery.

There are no subjective or constitutional symptoms. There may be pain from rubbing and forcibly removing the horny plugs.

Etiology and Pathology.—The condition is excessively rare. Brooke described nine cases occurring in one family; Graham Little[1] has had a group of three cases in one family; Barbe[2] two cases in one family; and Elliot[3] has reported a case. Previously to Brooke the same affection was perhaps described by Morrow,[4] and Leloir and Vidal, and Brooke regards as the same affection various other cases of follicular keratosis previously described.

The family groups of cases strongly suggest contagion. In one of Brooke's groups the condition began first in one of the children, and in the course of a few weeks spread to six of the seven children. Most of the cases have been in children, but not all of them. There has been no evidence of heredity in the cases. Brooke's cases occurred in vigorous, clean people of the working class.

Histologically the lesions are very like those of keratosis follicularis, but without the presence of psorospermlike bodies.

Diagnosis.—It resembles somewhat keratosis follicularis and ichthyosis. There is entire absence of the papillomatous new growths, of odor, and of greasiness of the skin found in keratosis follicularis, and there may be evidence of contagion. It is not a congenital or hereditary affection like ichthyosis, and unlike ichthyosis tends to spontaneous recovery and is amenable to treatment.

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[1] *Brit. Jour. Derm.*, vol. xiii, 1901, p. 417.
[2] *Annales*, vol. ii, 1901, pp. 422 and 535.
[3] *Jour. Cut. Dis.*, vol. xii, 1894, p. 362.
[4] *Ibid.*, vol. iv, 1886.

Prognosis and Treatment.—The disease yields without much difficulty to bathing and persistent inunctions of green soap.

XVI.—ACANTHOSIS NIGRICANS[1]

SYNONYMS: *Papillary and Pigmentary Dystrophy, Keratosis nigricans.*

Acanthosis nigricans is a disease characterized by the development of more or less symmetrically distributed areas of pigmentation and warty hypertrophy of the skin usually associated with visceral carcinoma.

The condition was originally described by Pollitzer and Janovsky in the same number of the "International Atlas of Rare Skin Diseases," and cases have since been reported by Darier, Hallopeau, Morris, Kuznitsky, Neumann, Spietschka, Boeck, Roberts, Dyer, and others. About thirty cases have now been described.

Symptomatology.—The affection begins rapidly or slowly. In Pollitzer's case it began at the commissures of the lips, and in eight weeks reached its full development. The lesions of the disease consist of patches of brownish pigmentation over the areas of distribution and of warty papillomatous growths of dirty brown to blackish color. The skin, in general, in the areas of distribution is thickened with exaggeration of the natural lines, and in addition there are papillomatous, horny, wartlike growths which may be thickly set and discrete or agglutinated into blackish, wartlike masses closely resembling pigmented verrucose nævus.

The lesions show a distinct predilection for the face and neck, the backs of the hands, and for deep folds of skin and for the commissures of the mouth. In Pollitzer's case the hands and the back of the neck were uniformly covered by the closely set, black, warty, papillomatous masses, so that they were of a diffuse dark-brown color. At either commissure of the lips, in his case, there was a thumb-nail-sized warty growth made up of agglutinated horny papillæ, while over the vermilion border of the lips and on the upper lip and chin were innumerable closely set but discrete minute blackish horny papules. The accumulation of the lesions into blackish, warty masses is common in all of the deep folds of the skin. On the palms and soles the process shows as keratoses, usually without pigmentation. Between these areas of involvement the skin may be pigmented or normal. Usually the oral mucous membrane is markedly involved, and covered by papillomatous granulations closely resembling acuminate condylomata. There are usually nutritional disturbances of the hair and nails, with partial or complete loss of the hair. Darier has observed the development of freckles, seborrhœic warts, and telangiectases along with the characteristic lesions.

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[1] Pollitzer, "International Atlas of Rare Skin Diseases," 1890, Plate X.—Janovsky, *ibid.*, Plate XI.—Darier, *Bull. Soc. Derm. et Syph.*, 1893, p. 421; *Annales*, 1893, p. 865, and 1895, p. 97.—Dyer, *New Orleans Med. and Surg. Jour.*, 1898, vol. li, p. 201.—Burmeister, *Archiv*, 1899, vol. xlvii, p. 343 (review and bibliography).—Couillaud, *Gaz. des Hopitaux*, 1897, p. 413 (review and bibliography).—Abstracts and Reviews of Papers, *Jour. Cut. Dis.*, 1897, p. 588, and 1899, p. 97.—Hodara, *Monat.*, 1905, vol. xl.—Heuss, *Monat.*, 1901, Bd. xxxii.—Pawlow, *Russ. Zeitschr. f. Derm. u. ven. Krankheiten*, Bd. i, Heft. 8.—Grosz, *Wien. klin. Wochenschr.*, 1902, No. 5.—Rille, *Heilkunde*, November, 1901.—Hess, *Münch. med. Wochenschr.*, 1903.—Grouven and Fischer, *Archiv*, 1904, Bd. 70.

After its full development the condition may vary in its intensity, but pursues a persistent course, which usually ends, in the course of months to several years, in the death of the patient from carcinoma. The lesions are without subjective symptoms. The constitutional symptoms are those of the cancerous cachexia.

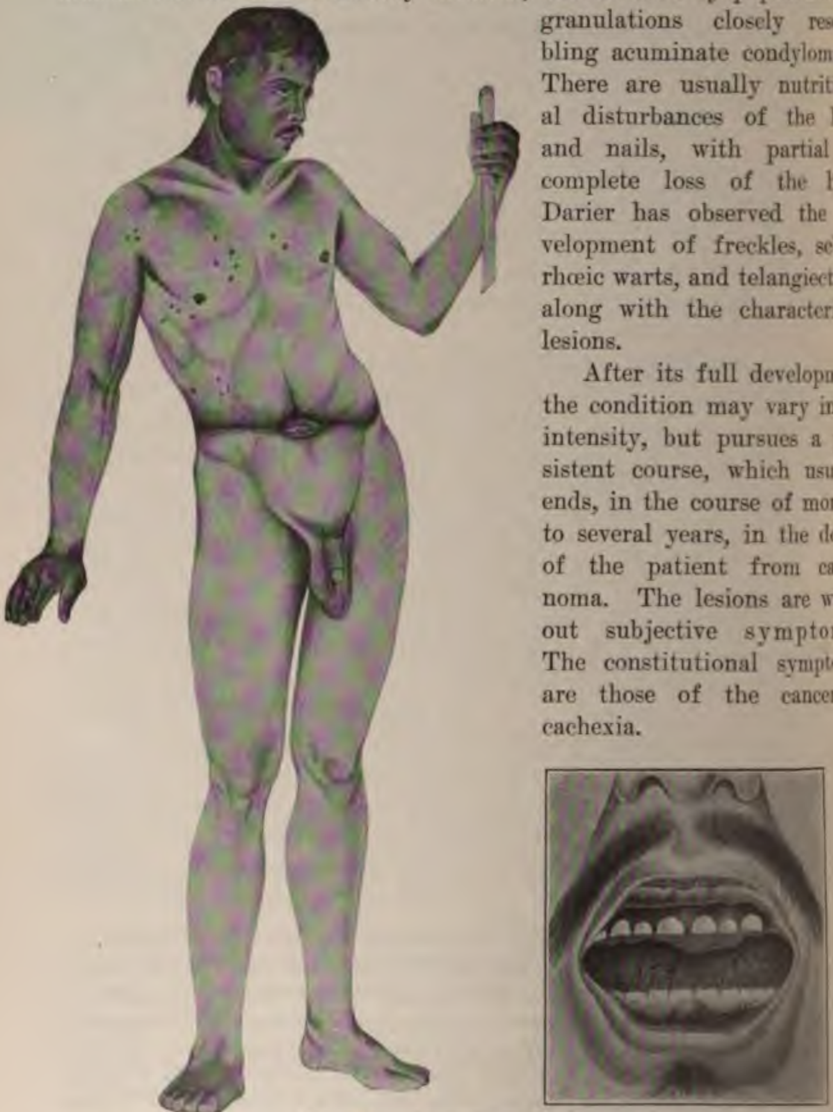

Fig. 245.—Acanthosis Nigricans.
(Photographic reproduction from Janovski's colored illustration, "International Atlas of Rare Skin Diseases.")

Etiology and Pathology.—The disease has been, in a large proportion of cases, associated with carcinoma, and presents some analogies o the increased freckling and pigmentation which are not infrequently een in association with visceral carcinoma. Darier's conclusions concerning it are as follows: "The disease is a syndrome dependent upon abdominal carcinosis, and characterized (I) from a clinical viewpoint by (1) a papillary hypertrophy and a cutaneous pigmentation having an essentially regional character; (2) a papillary hypertrophy of the muous membrane; (3) a dystrophy of the hair and nails; (4) absence of desquamation; (5) existence of a cachexia; (II) from a pathologic tandpoint, by carcinomatous degeneration of the abdominal organs; (III) histologically, by lesions of hypertrophy and pigmentation in the rete and corium." Other cases have been associated with occupations nvolving exposure to high temperatures.

Diagnosis.—The syndrome is characteristic. From ichthyosis it is readily distinguished by the fact that ichthyosis is a congenital condition, and does not affect the mucous membranes. In keratosis follicularis the hyperkeratosis occurs around the hair follicles, there is no disturbance of the general health, and microscopically there are present the psorospermlike bodies.

Prognosis and Treatment.—Treatment has been without avail.

XVII.—SCLERODERMA [1]

Scleroderma is a term applied to several conditions characterized by firm, chronic, noninflammatory infiltration of the skin. It occurs n two forms: *diffuse symmetrical scleroderma* and circumscribed scleroderma or *morphea.*

As to whether these two forms are distinct affections, there is difference of opinion. The clinical picture of circumscribed scleroderma or morphea is very distinct, and differs greatly from that of diffuse scleroderma. Morphea occurs in peculiar patches of yellowish, ivorylike nduration; diffuse scleroderma is merely a dense infiltration of the skin, and the induration is not nearly so distinctive in appearance as that of morphea. There are, however, certain cases in which diffuse areas of scleroderma and typical patches of morphea occur together. These cases indicate that at least some of the diffuse cases of scleroderma are identical in character with morphea. To my mind all the facts indicate that morphea is a distinct pathological entity, while diffuse scleroderma is a clinical entity, probably of varied pathology. Morphea is not very rare, and is not a grave affection. Diffuse scleroderma is very rare and of serious importance.

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[1] Krzysztalowicz, *Monat.*, 1906, vol. xlii, p. 143 (histology).—Towle, *Boston Med. and Surg. Jour.*, December 8, 1904.—Lewin and Heller, "Die Sklerodermie," Berlin, 1895 (review of the subject).—Osler, *Jour. Cut. Dis.*, 1898, pp. 49 and 127.—Dercum, *Jour. Nerv. and Ment. Dis.*, July, 1896, and October, 1898.—Fox, Colcott, *Brit. Jour. Derm.*, 1892, p. 101 (historical review).—Harbinson, *Brit. Med. Jour.*, January 16, 1904, "Scleroderma in Connection with Raynaud's and Addison's Diseases."

XVIII.—DIFFUSE SYMMETRICAL SCLERODERMA

SYNONYMS: *Hidebound Disease, Sclerema or Sclerema adultorum, Scleriasis, Dermato-sclerosis, Chorionitis, Sclerostenosis.*

Diffuse scleroderma is an affection in which large, symmetrical areas of skin become diffusely indurated and stiff, it may be without other changes. The disease is not strictly one of the skin, usually involving also the tissues beneath to a greater or less extent.

It occurs in two forms—the *infiltrated* and the *œdematous and atrophic.* Both forms are very rare.

Symptomatology. Infiltrated Form.—Either with or without premonitory symptoms, there develops rapidly induration of the skin and subcutaneous tissues. This may suddenly appear and involve a smaller or larger part of the body, or it may first appear as a more or less circumscribed area which gradually spreads. The distinct characteristic change is the infiltration and rigidity of the skin. It becomes thickened and tense, but the infiltration is firm and cannot be pitted like œdematous skin. The normal lines are in large part smoothed out, the skin is smooth and shiny and waxy in appearance, and the involved parts have an unnatural regularity of contour. The color may remain normal or be yellowish or grayish. There may be erythematous patches and groups of telangiectases, and there is frequently irregular pigmentation of the areas, usually of light brown, in extreme cases almost black. The borders of the patches are indefinite, and gradually merge into the normal skin. At times there is a zone of dilated vessels at the borders. The temperature of the affected area may be reduced one or two degrees or may be normal. There may be interference with the secretion of sweat and loss of hair, as well as nutritional changes in the nails when the extremities are affected. The infiltration usually involves the subcutaneous tissues, and may invade the underlying muscles, and the interference with mobility of the parts thus produced is the most characteristic feature of the condition.

The infiltration is always symmetrical in distribution. It frequently begins with stiffness in the back of the neck, chest, shoulders and arms, and the sites of predilection are the upper third of the body, the face, head and neck, and the arms. Next after these sites the legs are most frequently affected, and any part of the body may be involved, the s and soles, however, escaping except in the rarest instances. It spread to the mouth, pharynx, larynx, œsophagus, and vagina. As sult of the interference with mobility the affected parts become more ess fixed. The face may become expressionless and waxlike, with t interference with the movements of the lips and eyelids. When

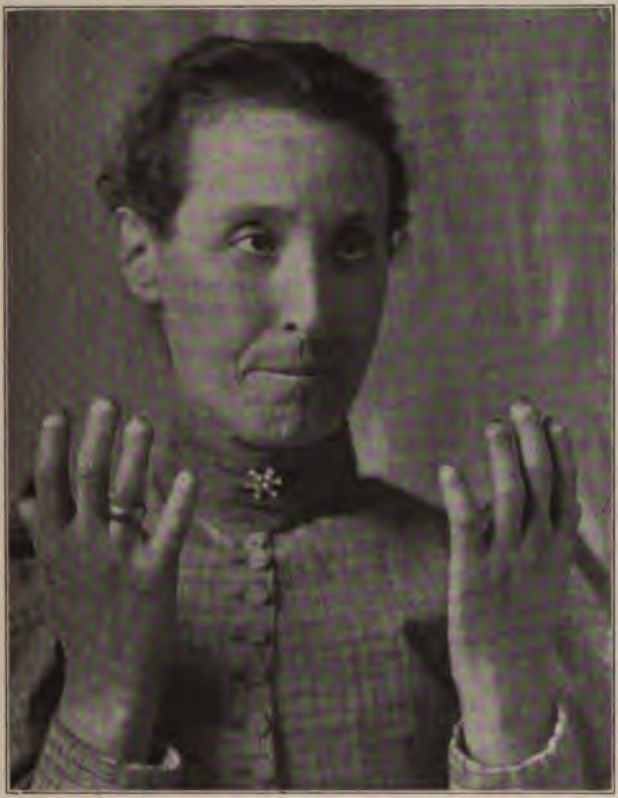

FIG. 246.—SCLERODERMA AND SCLERODACTYLIA. (Pollitzer's collection.)

chest is involved it may be to a greater or less extent fixed, with ening of the breasts and, in extreme cases, great interference with movements of respiration. When the extremities are involved there be partial interference with motion or complete ankylosis of the ts; the ankylosis, however, is never bony, but due to the fibrous ractures. The smaller motions of the fingers and toes are particu- interfered with, and in extreme cases motion is completely pre- ed and the hands become flexed and talonlike.

Edematous Form.—In this form of scleroderma the condition begins n œdema of the parts rather than a firm induration. This œdema is not, however, the ordinary serous œdema, but is a firm, doughy œdema which pits under the finger "like lard in a bladder" (Wilson). After persisting for a longer or shorter time the œdema subsides into an infiltrated stage, and then passes into atrophy. According to Crocker, atrophy only follows the œdematous cases.

The atrophic stage of scleroderma is represented by wasting both of the skin and underlying tissues. The skin becomes thin, dry, and closely drawn over the underlying structures, and fissures and ulceration are common. Accompanying this there is wasting of the underlying connective tissue and muscles which greatly diminishes the bulk of the affected parts. The face becomes drawn and ghastly, the lips and nostrils shrunken, and the other features atrophic. The lids usually escape, but may be involved, and there may be corneal ulceration. The atrophy may also involve the tissues of the mouth. The process on the limbs greatly reduces their size; flexure and ankylosis of the joints occur, and distortion of the hands and feet—*sclerodactylia.*[1] In the atrophic lesions of the extremities ulcerations are particularly likely to occur.

Both Forms.—There may be no interference with the general health, but usually the patients are not of normal vigor. Complicating diseases are common. Acute rheumatism is the most frequent complication, and occasionally endo- and pericarditis occur. Muscular and neuralgic pains are frequent, and myositis and peripheral neuritis have often been observed. Subcutaneous nodules, especially over the bones, which are apparently of the same character as rheumatic nodules and disappear spontaneously, have been observed in a few cases by Hutchinson, Crocker, and others. Both enlarged and diminished thyroid glands have been found. Raynaud's disease is the commonest accompanying skin disturbance, and various other vasomotor disturbances have been noted, such as transitory swellings of the skin, factitious urticaria, and lupus erythematosus.

Ordinarily there are no subjective symptoms assignable to the skin. Occasionally, however, there is itching or tenderness of the surface, and either hyper- or hypæsthesia may exist.

The disease may develop either insidiously or rapidly. Its course is usually very chronic. According to Crocker, the infiltrated cases pursue a less persistent course than the œdematous and atrophic. In the infiltrated cases the infiltration may be slowly absorbed and the skin gradually restored to normal. The process may also be arrested in the atrophic form, but there is no hope of restoring the wasted parts. In many cases the disease pursues a progressive course. Finally the patients levelop rheumatoid pains and neuralgias, cachexia appears, and they lie from some visceral disease or from marasmus.

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[1] Weber, *Brit. Jour. Derm.*, February, 1901.

Etiology and Pathology.—Of the actual cause of the disease we mow nothing. It may develop without any assignable cause and withut any concomitant disturbance of the health. Exposure to wet and old has frequently been observed to precede its development. On the ther hand, a case has been reported by Pick following prolonged exosure to the sun. Raynaud's disease has preceded it in some cases. t has frequently been observed to follow infectious diseases, of which ie most common are acute rheumatism and erysipelas. Other condions with which it has been associated are tuberculosis, diphtheria, and arlet fever. A few cases have been noted to follow injuries.

It has been observed three times as frequently in women as in men, id usually occurs in early or middle adult life. The extreme limits its appearance are twelve months and seventy years. In a case served by Crocker in a child of twelve months, it pursued a rapid urse. There was some improvement within three weeks, and recovery ok place within a year.

Histologically the epidermis is practically normal, the only change ing pigmentation of the deeper layers. The vessels of the corium are rrounded by layers of cells, which by their pressure cause narrowing the lumina, and the vascular caliber is sometimes diminished by hyperastic changes in the intima and media. Collections of cells similar those about the vessels are present around the sweat and sebaceous ands and hair follicles, and in the subcutaneous fatty tissue. The iture and origin of these cells is undetermined; no other phenomena inflammation are present.

Throughout the corium the white fibrous and elastic tissues and ie muscular fibers are increased, probably secondarily to the vascular ianges. The increase is due mainly to hypertrophy of the pre-existing ssue, in less degree to new formation; a dense, compact mass results, which the lymph spaces have almost entirely disappeared. The glands, iir follicles, and subcutaneous fat all tend to atrophy and disappear ıder the pressure, which also further diminishes the caliber of the ssels.

The generally accepted theory of the pathogenesis of both diffuse id localized scleroderma attributes the vascular changes to a lesion defect in the nervous system. No constant or directly connected sion has been found.

Diagnosis.—The diffuse, waxlike infiltration of the skin, immobiling to a greater or less extent the involved parts, is characteristic. he only condition resembling it is sclerema neonatorum, but in this iere is coldness of the surface, and it is an affection of the newborn, while the earliest case of scleroderma recorded was in a child of twelve months. The condition might possibly be confused with general idiopathic atrophy of the skin, with xeroderma pigmentosum, and with *cancer en cuirasse.* The course of scleroderma, with a history of preceding induration in the atrophic cases, distinguishes the condition from primary atrophy of the skin. The characteristic features of xeroderma pigmentosum are so distinct as to leave no possibility of confusion when the condition is known. *Cancer en cuirasse* is carcinoma spreading along the subcutaneous lymphatics. It produces a diffuse infiltration which is more or less nodular, is mottled by pinkish to purplish lesions, and is accompanied by involvement of glands and rapid cachexia.

Prognosis.—The disease tends in many cases to spontaneously disappear. It rarely ends in less than twelve months, and may extend over several years. In the atrophic cases the patients usually succumb to marasmus or some intercurrent disease.

Treatment.—The indications for treatment are to protect the patient from cold, to build up the general health as far as possible, and to stimulate the circulation in the affected areas. The patient should dress and live so as to avoid chilling as far as possible, and residence in a warm climate is desirable. Cod-liver oil and iron are the most useful tonics, but other tonics may be indicated. Neither arsenic, mercury, potassium iodide, nor any other specific has proved of any benefit. To stimulate the skin, frequent warm shampooing, or massage with an oil is most beneficial, and should be persistently carried out. Galvanism may be of benefit. Thyroid extract has been tried in many cases, with success in a few.

XIX.—MORPHEA[1]

SYNONYMS: *Circumscribed scleroderma, Addison's Keloid.*

Morphea (Greek, *μορφή*, a figure) is a disease characterized by the occurrence of persistent yellowish, ivorylike areas of infiltrated skin surrounded by faint lilac borders.

Symptomatology.—Morphea may begin as hyperæmic, coin-sized patches, which may or may not at first be slightly infiltrated and puffy. Ordinarily in the course of several days the pinkish color fades out, and there is left the characteristic indurated central patch surrounded by a pinkish or lilac zone. Occasionally the primary hyperæmic stage has been prolonged for months before the typical lesions developed. In other cases the lesions appear primarily as typical ivory-white areas of induration with a pinkish border. At other times the lesions begin as small white, depressed, scarlike spots, which coalesce to patches. However they begin, the lesions are usually quickly trans- rmed into typical patches. These typical patches consist of areas, regular in both size and shape, of infiltrated, dense skin of character- ic yellowish old-ivory color, with a pink or lilac border. Occasion- y the color may be somewhat mottled by a fine network of minute od vessels beneath the surface. The surface is smooth and glistening, th partial or complete obliteration of the normal markings. It may ve the feel of normal skin, but is more frequently firm and dense, d cannot easily be picked up or rolled between the fingers. The center y be level or slightly depressed, and rarely from contraction of the tral areas the borders may show minute radiating corrugations. ound the central area there is a narrow faint violet or pinkish zone, duced by dilated blood vessels. This faint lilac zone is very charac- istic of the lesions, but occasionally may be absent. There is decrease absence of sweat and of hairs in the patches.

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[1] Zarubin, *Archiv*, 1901, vol. lv.

The lesions of morphea are ordinarily from the size of a coin to at of a hand, rarely larger. Usually they are more or less roundish oval patches, but there is no regularity in their shape. They may cur in herpetiform groups of small spots; again they occur in narrow reaks; at other times in linear bands, perhaps an inch or two wide d many inches in length. The ordinary patches are not adherent to e subjacent tissues. The band-form lesions are not infrequently ad- rent to the underlying tissues, and then show as depressed, smooth, arlike bands. When not adherent the band forms may be distinctly evated, and then resemble hypertrophic scars. The lesions are usually ngle or few; occasionally they are numerous. In the great majority cases they are distinctly unilateral and of irregular distribution, though rarely they are bilateral and symmetrical. Sometimes the sions occur in herpetiform groups and sometimes follow the course cutaneous nerves; the band-form lesions are apt to occur on the ex- emities in long lines parallel to the axes of the affected parts.

Morphea may occur anywhere, but the sites of predilection are the tremities—the legs more frequently than the arms, the breasts, the ad, face, and neck.

The course of morphea is prolonged and indefinite; rarely both evo- tion and involution may be rapid. After a patch reaches a certain ze it may remain stationary for months or years, and then gradually sappear with restoration of the skin to normal. Or the patch, after maining stationary, may increase either by spreading of the diffuse filtration at the border or by the formation of small white daughter ots, which enlarge and coalesce with the mother patch. The duration the affection as a whole is altogether uncertain. A lesion may persist r several years or the disease may be continued by the occasional evolution and involution of patches. Ordinarily morphea disappears without leaving any trace, but at times patches of morphea are followed by atrophy, not only of the skin, but of the subjacent tissues. Occasionally cases have been observed in which morphea on the face, in children, has been followed by arrested development and facial hemiatrophy. In a case under my care, when I first saw the young boy there was a patch of typical morphea on the cheek near and parallel to the nose. In the course of two years this had been followed by facial hemiatrophy.

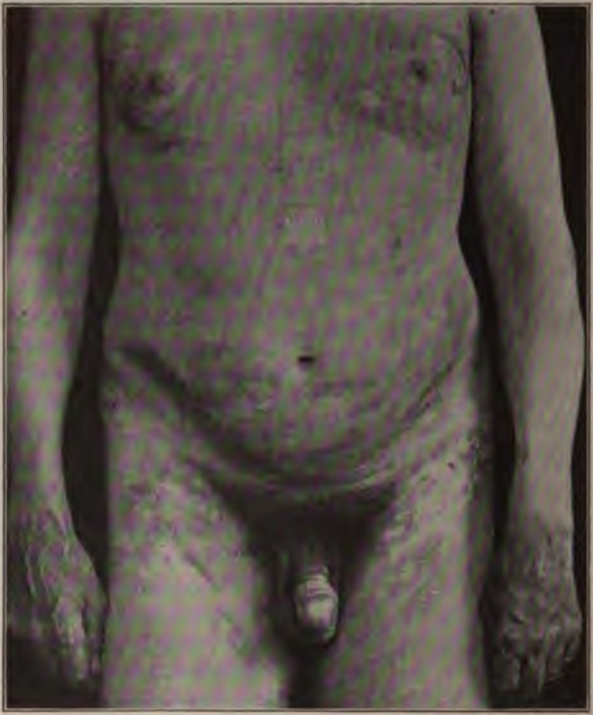

FIG. 247.—VERY EXTENSIVE MORPHEA. Dark areola of the lesions violaceous in life. (Author's photograph of Drs. Kanoky and Sutton's case.)

There are no characteristic subjective or constitutional disturbances. Not infrequently there is some itching or tingling in the patches.

Variations.—Numerous variations are occasionally observed from typical morphea.

Occasionally the patches are not of the characteristic yellowish ivory color, but may be pink or violaceous from the network of blood vessels, or they may show varying degrees of pigmentation from brownish to dark brown, or even dark green or black.

In some cases the lesions, while presenting the other characteristics of morphea, may be atrophic instead of infiltrated, occurring as thin, white, mottled, slightly depressed patches which are soft and pliable, but are surrounded by a lilac or pigmented zone. The identity of these patches with typical morphea is shown by the occurrence at times of both infiltrated and thinned lesions. The similarity of these atrophic cases to *white spot disease* should be borne in mind.

Along with the typical patches there may be white atrophic lines and ots, telangiectases, and areas of pigmentation. These may disappear r develop into typical lesions of morphea.

Occasionally ulceration occurs in the areas, and if it occurs in one sion it is likely to occur in the others. As Crocker suggests, consider- g the amount of blocking of the blood vessels the wonder is that the ceration is so rare. Sherwell and Morrow have also observed bullæ velop upon the lesions.

While all of these variations from the typical patches of morphea y occur, they are very rare, and seldom confuse the characteristic nical picture.

Etiology and Pathology.—The actual cause or causes of morphea, of diffuse scleroderma, are unknown. It is more common in women an in men, and there is apparently often a nervous element in the ses, the disease most frequently being seen in persons showing some idences of unstable nervous equilibrium. Anxiety, worry, shock, and her causes of nervous exhaustion are apparently in some cases definitely sociated with the development of the affection. In a small propor- n of cases the locations of the lesions seem to indicate the influence local irritation. Thus the exciting cause of some of the lesions seem be the garters, the friction of the shoes, the irritation of the breasts oduced by corsets, the friction of the neckbands of clothing. In other stances the lesions have followed wounds, and Barthélemy has reported case following x-ray exposures.

The histology is in general similar to that of the diffuse form. rocker describes collections of cells around the vessels, especially of e superficial plexus, which he considers primary and which by pressure roduce diminution of the vessel caliber, and consequent thrombosis; rombi occurred in many of the vessels, extending up into the papillæ. the later stages the most marked changes are the increase of fibrous ssue and pressure atrophy of the glands and follicles. Thus each spot the base of a cone whose blood supply has been cut off—i. e., is prac- cally a cutaneous infarct; collateral hyperæmia produces the surround- g violaceous zone.

Diagnosis.—Typical morphea is not readily confused with any other rmatosis. Leukoderma differs from it in that it is merely a loss pigment without changes in the texture of the skin. Scars and loids may at times closely resemble morphea. In scars there should obtainable a history of previous injury, and there is entire ab- nce of the peculiar lardaceous induration and of the lilac border of orphea. In keloid the lesions are more vascular and of greater ickness, and they have radiating processes extending from the main sion.

Prognosis.—The lesions ultimately disappear, usually without atrophy. Improvement may be rapid or slow and patches may last from two or three to twenty years or longer. According to Crocker, band forms are less favorable than the patch form of lesion.

Treatment.—The treatment of morphea is altogether unsatisfactory. As much as possible should be done on general principles in the way of symptomatic constitutional treatment.

Various methods of local treatment have been tried: galvanism, electrolysis, massage, and the injection of thiosinamin, as in the treatment of keloids. Of these measures, massage with a bland fat or oil offers the best prospect of benefit.

XX.—MIXED SCLERODERMA[1]

Rare cases have been reported by Addison, Gaskoin, Duckworth, and others of mixed diffuse scleroderma and morphea which showed the general characteristics and have followed the course of circumscribed scleroderma. The cases are especially interesting as showing the connecting link between morphea and diffuse scleroderma. Eddowes[2] has demonstrated a case presenting at the same time general alopecia, leukoderma, and mixed form of scleroderma, illustrating how the various hypertrophies and atrophies of the skin are related.

XXI.—WHITE SPOT DISEASE[3]

SYNONYM: *Westberg's Disease.*

In 1901 Westberg reported under this name a case occurring in a girl eleven years old, in which there were round and oval coin-sized or smaller white spots in the skin of the thorax. They were smooth, without desquamation or other changes in their surface, and, upon stretching the skin, stood out like neoplastic infiltrations. F. H. Montgomery demonstrated before the American Dermatological Association, in 1901, a similar case, and Johnston and Sherwell have reported a case in detail. One case in which the lesions consisted of a group of ten or twelve white atrophic spots over the pectoral muscle has occurred in my practice in the person of a woman aged about forty. The other cases have been in women or girls. Montgomery's case at the time of the demonstration was generally regarded as a peculiar form of morphea. The lesions are, I believe, lesions of morphea. Johnston regards them as a distinct affection.

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[1] Stowers, *Brit. Jour. Derm.*, 1905, p. 106.

[2] *Brit. Jour. Derm.*, 1899, p. 325.

[3] Westberg, *Monat.*, 1901, vol. xxxiii, p. 355.—Johnston and Sherwell, *Jour. Cut. Dis.*, 1903, p. 302.—F. H. Montgomery, *Trans. Am. Derm. Assn.*, 1906.

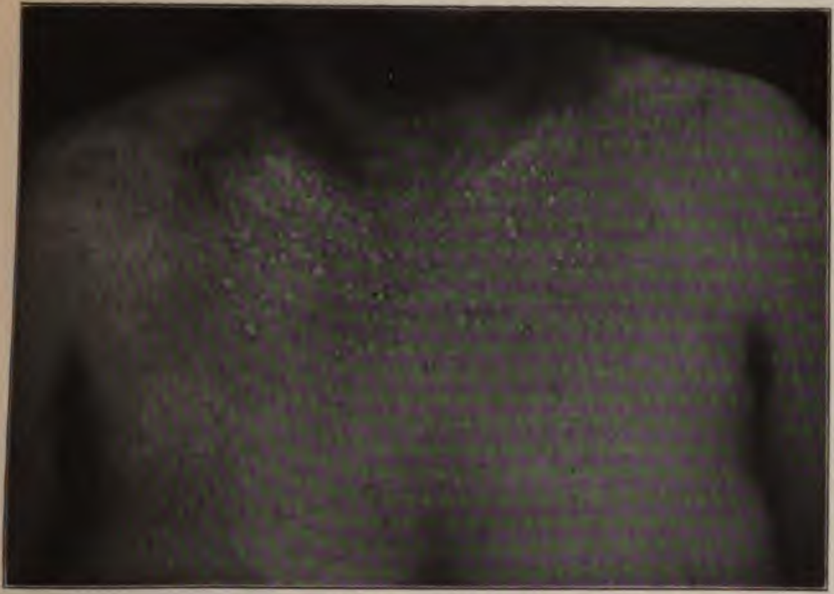

Fig. 248.

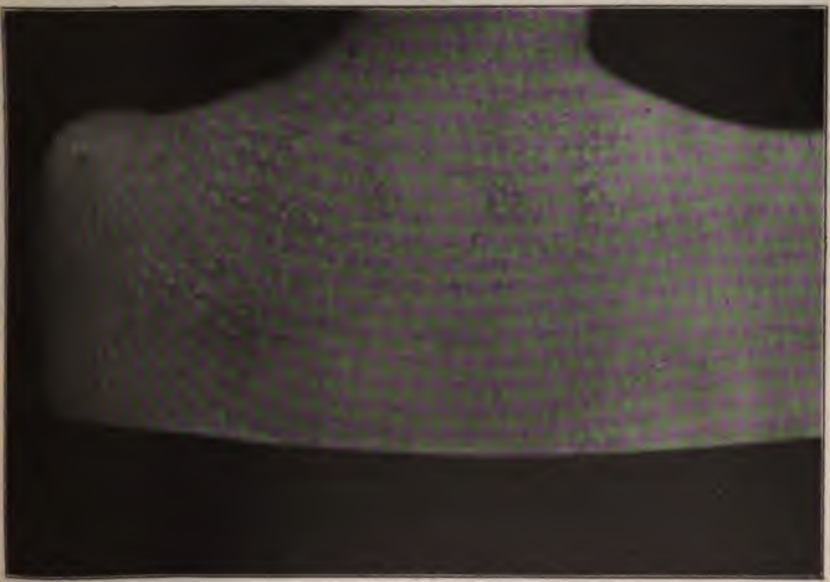

Fig. 248*a*.

Figs. 248 and 248*a*.—White-spot Disease. (Montgomery and Ormsby.)

XXII.—CUTIS PLICATA

Heller has described from one case a curious hardness of the skin which caused the normal furrows to be exaggerated and the skin to be thrown into folds, giving the appearance of an extreme degree of senile atrophy. The skin, however, was not atrophic. The epidermis was normal except for dilatation of the hair follicles, and the anomaly consisted of a dense thickening of the corium. The hands had a violaceous red color, such as is seen in laborers who are exposed to cold. There were no subjective symptoms, and the condition had existed since earliest childhood. The patient, an artisan, aged twenty-one, had sought advice for cardiac distress, but no disease of the heart or arteries was found. Heller refers to analogous cases seen by Waelsch and Hochsinger and Jadassohn. The description suggests a stage of induration which I have seen precede idiopathic atrophy of the skin.

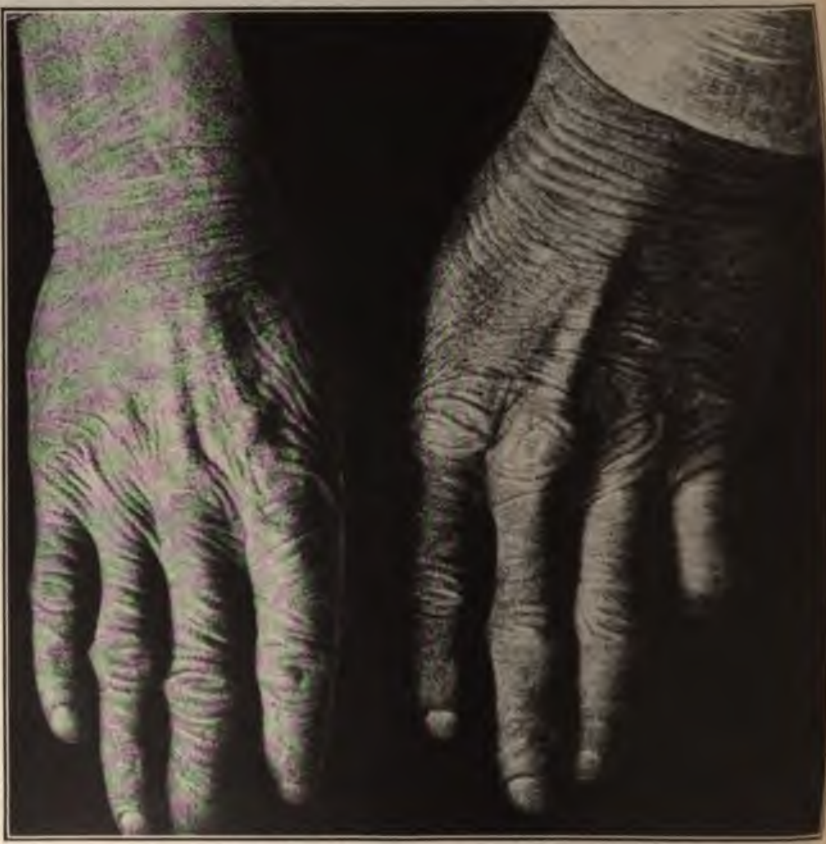

FIG. 249.—CUTIS PLICATA. (J. Heller.)

XXIII.—SCLEREMA NEONATORUM AND ŒDEMA NEONATORUM

These are two affections, developing at birth or within a few days after, which in their clinical manifestations closely resemble respectively the infiltrated forms of diffuse symmetrical scleroderma and the œdematous form of symmetrical scleroderma. Their peculiar feature, which distinguishes them from scleroderma, is their appearance at birth; and they are generally regarded on that ground and upon the differences in the histological pictures as distinct diseases. They are, however, excessively rare, and opportunities for studying them have been few, so that in my opinion it is not established that they are not at least closely related to scleroderma.

SCLEREMA NEONATORUM[1]

SYNONYM: *Scleroderma neonatorum.*

Sclerema neonatorum is a diffuse, usually universal, firm infiltration of the skin, occurring at or soon after birth.

Symptomatology.—The condition usually begins as an infiltration of the skin and subcutaneous tissues of the legs, which, spreading rapidly, usually becomes universal or almost so in the course of three or four days. The changes are those of a dense waxy infiltration of the skin. The skin is smooth and tense; at first whitish, it becomes later livid or mottled. It is firm and cannot be rolled between the fingers, and does not pit on pressure. The temperature is reduced and the surface cold and clammy. The infiltration extends to the subcutaneous tissues so that mobility of the parts is greatly lessened and the infant is converted into a stiffened, corpselike figure. It is unable to move, cannot nurse or nurses with difficulty, and respiration is interfered with. In some cases the infiltration does not become general, and in such cases recovery may take place rapidly or the infiltration remains stationary for a longer or shorter time. In the usual cases in which the condition quickly becomes universal the temperature remains subnormal, and the child dies in a few days or one or two weeks from lack of vitality, loss of heat, or, it may be, from intestinal disorders, pneumonia, or circulatory disturbances.

Etiology and Pathology.—The disease was first described in 1784 by Underwood, who regarded it as a result of bad hygiene and improper feeding. Parrot, who first drew a clear distinction between it and œdema neonatorum and whose studies established both affections as clinical entities, agreed in general with Underwood as to its causation. It is for the most part an affection of institutions, and is associated with depressing influences of various sorts, such as premature birth, cardiac weakness, atelectasis, diarrhea, and pneumonia.

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[1] Stillman, *Jour. A. M. A.*, April 25, 1903.—Taylor, *Brit. Med. Jour.*, April 21, 1900.—Browning, *Jour. Cut. Dis.*, 1900, vol. xviii.

The pathology is not clear. Langer regards it as due to solidification of the infantile fat on account of subnormal temperature. This does not correspond with the histological findings of Parrot, and is inconsistent with the fact that the condition may exist at birth. Parrot very plausibly regards the condition as due to desiccation of the tissues from diarrhea or other causes. He found, anatomically, great thinning of the skin throughout, except in the horny layer, with contraction of the blood vessels, especially of the papillary layer, and without true sclerosis or serous infiltration.

Prognosis and Treatment.—The exceptional cases which are only partial may recover. In other cases there is a fatal result. The treatment is the same as that for œdema neonatorum.

Œdema Neonatorum

Œdema neonatorum is a diffuse, rarely universal œdema of the skin occurring at or soon after birth.

Symptomatology.—Œdema neonatorum occurs as a more or less firm, doughy œdema involving the skin and subcutaneous tissues. The affected parts are swollen, tense, yellowish, white or livid, and cold. Pitting follows firm pressure, but the œdema is not the ordinary soft serous œdema. The œdema may be present at birth or appear during the first few days afterwards. Like other œdemas, it is most marked where the circulation is feeblest. It usually begins upon the lower parts of the legs, then rapidly appears upon the hands; it may, however, begin on the back or face. From the legs it spreads up over the thighs, and then involves the genitals and back. It is usually marked on the soles and palms and nates, which become reddened and hard. It rarely becomes universal. With the condition the child is drowsy, the circulation and respiration feeble, and the temperature subnormal, and death usually occurs in a few days.

In association with it there have been observed jaundice, erysipelas, pemphigoid eruptions, purpura, and gangrene of the skin, and in some of the cases there has been high fever.

Etiology and Pathology.—Among contributory or actual causes of the condition are premature birth, cardiac weakness, atelectasis, insufficient nourishment of the mother, and exposure of the infant to cold immediately after birth. The condition has been seen in association with nephritis, hereditary syphilis, erysipelas, and other infectious diseases.

The condition is generally regarded as similar to other œdemas due to feeble or obstructed circulation. Ballantyne regards it as of renal, cardiac, or pulmonary origin. Thrombi have been found in the femoral veins in one case. Anatomically there is serous effusion into the connective tissue, with increase in density of the fat.

Diagnosis.—The characteristic difference between sclerema neonatorum and œdema neonatorum is that in œdema neonatorum there is œdema of the tissues, which pits more or less readily upon pressure and has the usual features of œdema more or less marked, while in sclerema neonatorum the infiltration of the tissues is hard, not serous. Œdema neonatorum is also usually a less general affection, and in it there is much less stiffening of the tissues and interference with mobility.

Prognosis and Treatment.—The cases are usually fatal, but the prospect is not so hopeless as in sclerema and the course not quite so rapid. Indications for treatment are to supply heat and nourishment. The child must be kept warm artificially, best in an incubator, and must be fed through a tube passed either by the mouth or nose into the pharynx. Friction of the surface with oil in the direction of the venous flow may help in the establishment of the circulation.

XXIV.—ELEPHANTIASIS[1]

SYNONYMS: *Elephantiasis Arabum, Pachydermia, Barbadoes Leg, Elephantiasis indica.*

Elephantiasis is an hypertrophy of the skin and subcutaneous tissue produced by local circulatory disturbances. It usually involves not only the skin but the underlying tissues, but is confined to a member or region of the body.

Elephantiasis, as it occurs in tropical countries in connection with filaria sanguinis hominis, is a specific affection due to lymphatic obstruction by filaria. Sporadic elephantiasis is a symptom and not a specific pathological entity. It may result from any pathological process causing lymphatic obstruction.

Symptomatology.—The characteristic change of elephantiasis is enlargement of a region or member of the body. This enlargement is a true hypertrophy of the tissues. Unless there is superimposed serous œdema—which is an accidental complication that may involve an elephantiasic part as well as a normal part—it does not pit on pressure.

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[1] Manson, "Tropical Diseases," London, 1898.—Mastin, "Filaria Sanguinis Hominis in the United States," *Annals of Surgery*, November, 1888.—Hyde, Morrow's "System," vol. iii, p. 451 (full bibliography).

Congenital Elephantiasis: Busey, "Congenital Occlusion and Dilatation of Lymph Channels," New York, 1878.—Jopson, *Arch. Pediatrics*, 1898.

The tissue is of normal or of increased firmness. In the simplest forms of the affection the clinical manifestations are simply those of enlargement of the part, the skin remaining unchanged. In more exaggerated forms, particularly as they occur on the extremities, the skin becomes hard and sclerotic, there may be in addition papillary hypertrophy, particularly on the leg and foot, producing warty growth of the tissues; this may be unaccompanied by marked changes in the epithelium, but there is usually also **epithelial overgrowth** with the production of roughened, horny, verrucose skin, at least in circumscribed areas. When this condition becomes established fissures, eczema, or even ulcers are very apt to occur. This is particularly the case on the legs, where the inflammatory symptoms are the result in part of the stasis produced by gravity and in part by the increased vulnerability of the hypertrophied skin.

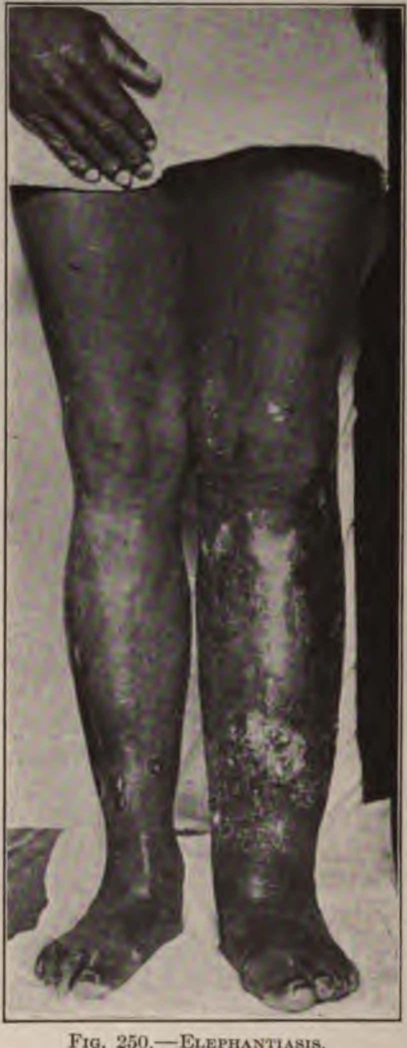

FIG. 250.—ELEPHANTIASIS. (Author's collection.)

The parts most frequently affected by elephantiasis are the legs, the genitals, the face, and the arms. It is far most common in the legs, and the right leg is said to be more frequently involved than the left. It may be limited to the foot and ankle. In the sporadic cases one leg is usually involved, but in the endemic variety it often affects both legs. When the genitals are involved, it may involve both scrotum and penis, but more frequently affects the scrotum chiefly. In women it may involve all of the external genitals, but is usually confined to the labia majora. In the leg and genital cases the inguinal lymphatics are usually enlarged. On the face it produces a diffuse swelling which strikingly resembles true œdema, except that there is no pitting on ressure. This form of elephantiasis, which results from repeated ttacks of erysipelas, is the so-called *solid* or *persistent œdema* of the ace. There is no limit to the size which a part affected by elephantiasis ay attain. Ordinarily the en- argement is only sufficient to nterfere to a moderate extent ith movement; in extreme ases the legs or scrotum or abia may become so enlarged hat from their very weight ovement becomes impossible. or example, Schwimmer re- ers to a scrotal tumor of 110 ounds and Manson to one of 24 pounds.

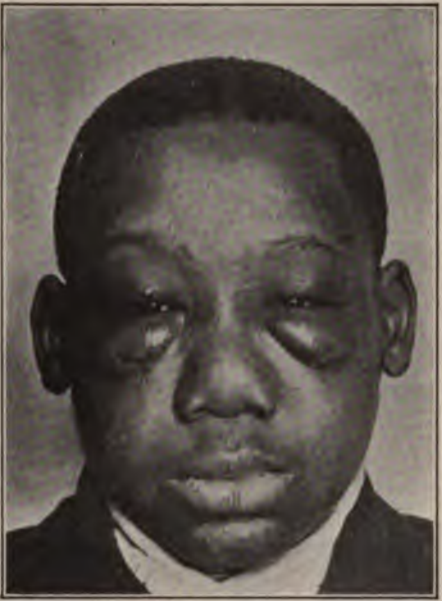

FIG. 250*a*.—ELEPHANTIASIS FOLLOWING NUMEROUS ATTACKS OF ERYSIPELAS. (Author's collection.)

The development of ele- hantiasis is always slow, its xtreme manifestations appear- ng only after months or years. s the condition is a sequel al- ays of some other pathological rocess, its development is pre- eded by the symptoms of the rimary affection. Nearly al- ays there is a history of recur- ent attacks of lymphangitis in he affected area. As the first manifestation of the condition there may be ersistent œdema of the part, which in the course of time is supplanted y connective-tissue hyperplasia.

Lymph-scrotum is elephantiasis of the scrotum accompanied by vari- ose lymphatics, from which upon rupture or puncture there is an exu- ation of pinkish or milky lymph. It is due to filaria, and is often ccompanied by hæmaturia and chyluria.

Elephantiasis telangiectodes is hypertrophy of parts accompanied by nlargement of the blood vessels. It is usually a congenital affection, nd according to Virchow is due to the overnutrition of the parts as a onsequence of abnormal vascularity. In addition to the usual enlarge- ent there is, as a result of the increased vascularity, more or less eddening of the surface. The condition is ordinarily congenital, but errill Ricketts[1] has reported a case involving the lower part of the ace which developed in adult life.

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[1] *Jour. Cut. Dis.*, 1889.

Etiology and Pathology.—Elephantiasis may be produced by any condition that causes persistent obstruction of the lymphatics. The endemic form of elephantiasis is produced primarily by the blocking of the lymph trunks by the filaria, and of the smaller lymphatics by the embryos. A secondary factor is probably the inflammation excited by the presence of the parasites. The sporadic cases are most frequently due to repeated attacks of erysipelas or of inflammatory processes produced by pyogenic streptococci. On the legs it not infrequently follows persistent long-standing cases of eczema, and it may follow lupous, syphilitic, and varicose ulcers. It may also be produced by the mechanical obstruction caused by tumors and scars, especially after removal of the inguinal glands. In some cases it is congenital as the result of congenital occlusion of the lymph channels.

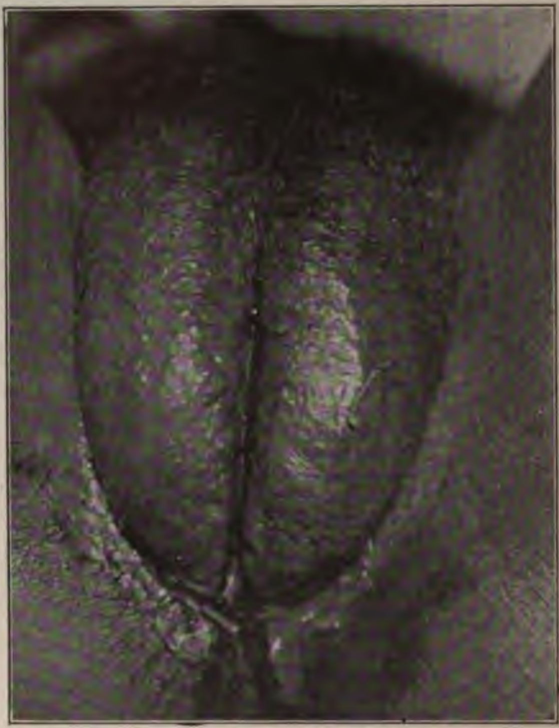

FIG. 251.—ELEPHANTIASIS VULVÆ IN NEGRO. (Author's collection.)

Sporadic elephantiasis is rather uncommon, but is by no means a rare condition. In endemic districts elephantiasis is common. It is about three times as common in men as in women. Following the usual greater susceptibility of dark races, particularly negroes, to fibrous tissue growths, it is much commoner in the dark races. The majority of the cases seen in America are in negroes.

However elephantiasis is produced, the pathological changes are the same, and consist of cell proliferation and hypertrophy involving, to a greater or less extent, all of the tissues of the part. The connective-tissue hypertrophy is the most characteristic feature, and converts the part into a dense fibrous structure in which there are usually found ilated lymphatics or lymph pockets from which lymph exudes upon cross ction. There is great thickening of the corium and matting together f its various layers so that their typical arrangement is completely disrganized. The epidermis may be normal or hypertrophied. The sweat nd sebaceous glands and the hair follicles may be unchanged, or may be estroyed in the sclerosis of the tissues. The sclerosis may result in fatty egeneration and atrophy of the muscles, but the bones are not affected.

Diagnosis.—The diagnosis of elephantiasis is a matter of no diffiulty. From acromegaly it is distinguished by the fact that in acroegaly there is symmetrical enlargement of the face, feet, and hands, hich is in no way associated with evidences or history of lymphatic bstruction or of recurrent attacks of inflammation, and in which there s marked enlargement of the bones as well as of the soft parts. In he thickening of the skin of the face in myxœdema, the thickening is ot nearly so great as in elephantiasis, and is accompanied by other haracteristic symptoms of myxœdema.

Prognosis and Treatment.—Elephantiasis cannot be relieved exept by surgical removal. Its course can be checked if the cause can e removed. In the cases produced by filaria the treatment is for that ondition. In the sporadic cases the treatment consists in the usual easures for the prevention of the recurrent attacks of erysipelas and n the usual treatment of the ulcers, the chronic eczemas, and the other esions which are the causes of the condition. In treatment of elehantiasis of the legs there is often much improvement from the cure f the inflammatory condition which accompanies it. An important actor in the treatment of elephantiasis of the legs is support of the irculation. This is best obtained by rest in bed, especially during xacerbations of the complications. Next to this is support by bandages. n addition to the benefit derived from bandages for the inflammatory omplications, considerable benefit may be obtained from the compresion of the parts by the continuous application of rubber or other ompressive bandages. Another measure sometimes employed is the ligaure of the main artery of the leg, which is usually accompanied, howver, by only temporary improvement. Elephantiasis of the genitals s best treated by ablation of the hypertrophied tissues. The results re usually satisfactory and permanent.

XXV.—DERMATOLYSIS

SYNONYMS: *Loose Skin, Cutis laxa, Cutis pendula.*

The term dermatolysis is applied to the condition in which the skin nd subcutaneous connective tissue become hypertrophied and lax, and end to hang in loose folds.

The condition is analogous to that seen normally in certain of the lower animals, like hounds, in which the skin can easily be picked up in the hands in large folds or hangs down in loose folds under the jaws and in the flanks.

Symptomatology.—In dermatolysis the skin is thickened, as a whole; and, as the follicles usually participate in the process, they are dilated and gaping. There may be comedones in the dilated sebaceous follicles, and the skin is usually greasy and soft. There may or may not be increased elasticity of the skin. There may be increased pigmentation.

The degree of the condition may only amount to an increased looseness of the skin, so that it can be picked up easily, or the condition may be so marked that the skin will hang in pendulous folds. It is usually circumscribed and confined to a few areas, but may be manifested extensively. It most frequently affects the skin of the face, especially the eyelids, the neck, the chest, the abdomen, and the genitals. There are no disturbances of sensation and no subjective symptoms.

Etiology and Pathology.—The etiology of the condition is obscure. It may be congenital or acquired; it may be hereditary; it may start at the site of an injury, or it may develop without any discoverable reason.

According to Duhring's findings, the changes in the skin consist chiefly in increase of the fibrous bundles and of the fat of the subcutaneous tissue and corium.

Diagnosis.—The condition has to be distinguished from fibroma pendulum. In fibroma pendulum the tumors are altogether irregular in distribution, entirely independent of the normal areas of lax skin.

Prognosis and Treatment.—There is no tendency for the spontaneous disappearance of the affection. Treatment, when necessary, is surgical.

XXVI.—ELASTIC SKIN[1]

SYNONYM: *Cutis elastica.*

This is a rare condition in which the skin, without other changes, possesses abnormal elasticity. The ordinary examples of the condition are the "india-rubber" men who exhibit themselves as freaks. The condition was accurately described as long ago as 1723 by Daniel Turner in the second edition of his "Diseases of the Skin." Turner called attention to the fact that the abnormal elasticity was confined to one

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[1] Duhring, *Med. News*, vol. xliii, 1883.—Kopp, *Münch. med. Wochenschr.*, 1888.—Du Mesnil, *Verhandl. der physic. med. Gesellsch. in Würzburg*, vol. xxiv, 1891.—Unna's "Histopathology," p. 984.

e, a fact which has been observed in other cases. The abnormal
sticity may be universal or limited to certain parts. It may be so
reme that the skin can be pulled out in thin folds, literally like india
ber, so that the patient "would draw the Skin of his Chin down
his Breast like a Beard, and presently put it upwards to the Top of
Head, hiding both his Eyes therewith; After which the same would
urn orderly and equally to its proper Place, lying smooth as in any
er Person" (Turner).

The condition is apparently dependent upon a peculiarly loose ar-
ngement of the connective-tissue bundles of the subcutaneous con-
ctive tissue which permits of unusual extension.

SECTION X

ATROPHIES

Synonyms: *Atrophia cutis, Atrophoderma.*

Atrophy of the skin may be either quantitative or qualitative; that is, it may be due simply to diminution in the bulk of the tissues of the skin without qualitative changes or it may be accompanied by degenerative changes in one or more of the elementary tissues. This distinction is in part academic, for it is not always possible to tell where simple diminution in bulk ceases and where degenerative changes begin—e. g., in senile atrophy of the skin the principal change is a simple quantitative atrophy; at the same time there are frequently degenerative changes which manifest themselves in areas of pigmentation, senile keratoses, and, it may be, in malignant growths.

Atrophy of the skin occurs symptomatically in the course of many dermatoses, like lupus erythematosus, nonulcerative syphilides, pityriasis rubra of Hebra, scleroderma, and after various ulcerative lesions (*symptomatic atrophy*). In a few conditions it is the essential pathological process (*idiopathic atrophy*).

The atrophies of the skin may be classified as follows:

I. Idiopathic atrophy:
    (1) Diffuse,
        (*a*) Senile atrophy.
        (*b*) Xeroderma pigmentosum.
        (*c*) Atrophoderma albidum (Crocker).
        (*d*) Diffuse idiopathic atrophy.
    (2) Circumscribed.
        (*a*) Kraurosis.
        (*b*) Striæ et maculæ atrophicæ.
        (*c*) Multiple tumorlike new growths.

Symptomatic atrophy:

(1) Neuritic,
  (*a*) Glossy skin.

(2) After various skin diseases,
  (*a*) Lupus erythematosus.
  (*b*) Scleroderma.
  (*c*) Pityriasis rubra of Hebra.
  (*d*) Favus.
  (*e*) After various ulcerative lesions, like syphilitic and lupous ulcers, etc.

The atrophies of the skin which are symptomatic of various derma- ses are taken up with the different diseases. There remain to be nsidered here the idiopathic atrophies and the atrophy following nerve ions.

There are also included in this group three trophic diseases which e not essentially atrophies:

(1) Perforating ulcer.
(2) Ainhum.
(3) Syringomyelia.

I.—SENILE ATROPHY OF THE SKIN

Synonyms: *Atrophia cutis senilis, Atrophoderma senile.*

Symptomatology.—The changes occurring in the skin in old age due chiefly to the loss of subcutaneous fat and the shrinking of the ium and epidermis. The changes in appearance of the skin in old are familiar. The skin becomes gradually drier, harsher in tex- e, and wrinkled. The pink and white tints are lost, and the color omes tinged with muddy yellow. There is a tendency to the forma- ı of pigmented spots, which frequently develop into the thickened, wnish, or blackish warty lesions that have been considered under ıile Keratoses. Such senile warts may persist indefinitely as such, may undergo degeneration into epitheliomata. Mingled with the ile keratoses there may be small clusters of dilated blood vessels ch show here and there as red spots in the skin; and atrophic white ts may also develop.

The senile atrophic changes are most marked on the face, neck, and ıds, although they occur to a greater or less extent over the entire face. The appendages share in the atrophy. In addition to the ıning or complete loss of hair upon the scalp, the lanugo hairs also ome thinner and less vigorous. There is some atrophy of the sebaceous glands, and diminution in the amount of sebum and sweat, although anatomical evidences of atrophy of the sweat glands is usually lacking. As a result of the failure of the secretions the skin is dry, and there is apt to be slight, branny desquamation. Late in the course of senile atrophy of the skin there is frequently itching, which may become distressing. This is considered under Senile Pruritus.

Etiology and Pathology.—The development of senile changes in the skin is usually very slow. They are rarely manifest before forty, but the age at which they become marked varies greatly in individuals. As is well known, certain individuals show wrinkling of the skin much more markedly than others, and the other changes in the senile skin differ correspondingly in different individuals and families. As a rule, the senile changes in the skin are most marked in those who are thin and have little subcutaneous fat. The individuals who have a thick layer of panniculus adiposus, who perspire freely and have an unctuous skin, show senile changes in the skin least distinctly.

Anatomically the changes consist in thinning of the epidermis and corium and shrinkage of the subcutaneous fat. The epidermis may be reduced to a thin, wavy line. The papillæ are flattened; the connective-tissue bundles of the corium are shrunken. The blood vessels in some parts, are obliterated, in others dilated. Pigment granules are found in the corium and, it may be, in the walls of the blood vessels.

Treatment.—Nothing can be done to prevent the senile thinning of the skin, but the dryness and harshness may be in part prevented and in part counteracted by bathing and the use of emollient applications. The treatment of senile keratoses has already been considered.

II.—XERODERMA PIGMENTOSUM[1]

SYNONYMS: *Atrophodermia pigmentosa* (Crocker), *Angioma pigmentosum atrophicum* (Taylor), *Dermatosus Kaposi* (Vidal), *Liodermia essentialis cum melanosi et telangiectasia* (Neisser), *Melanosis lenticularis progressiva* (Pick).

Xeroderma pigmentosum is a disease of the skin, usually beginning in early childhood and occurring in family groups, characterized at first by the development of an abundant crop of freckles accompanied by atrophic changes in the skin and later by the appearance of malignant growths. The disease was first described by Kaposi in 1870, and it is nately extremely rare; but it is so striking that the cases rarely recognition, and more than one hundred cases are on record.

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[1] Hebra, vol. iii.—Duhring, *Amer. Jour. Med. Sci.*, October, 1878.—Kaposi, *Wien. med. Jahrbücher*, 1882, p. 619, and *Wien. med. Wochenschr.*, 1885, p. 1334. —J. C. White, *Jour. Cutan. Dis.*, 1885, p. 353.—Taylor, *Med. Record*, March 10, 1888.—Brayton, *Jour. Cutan. Dis.*, 1892, p. 129, and *Jour. Amer. Med. Assoc.*, April 29, 1899.—Hutchins, *Jour. Cutan. Dis.*, 1893, p. 402.—Bronson, *Jour. Cutan. Dis.*, 1899, p. 572.—A. H. Bowen, *Jour. Amer. Med. Assoc.* (1 case), April 29, 1899.

ymptomatology.—Xeroderma pigmentosum is in its manifesta- practically the same as the most extreme examples of senile atrophy e skin, with an exag- on of the tendency to evelopment of cutane- arcinoma. It is ap- tly a congenital defect e skin, which results undergoing the most ne changes of senile hy in a few years.

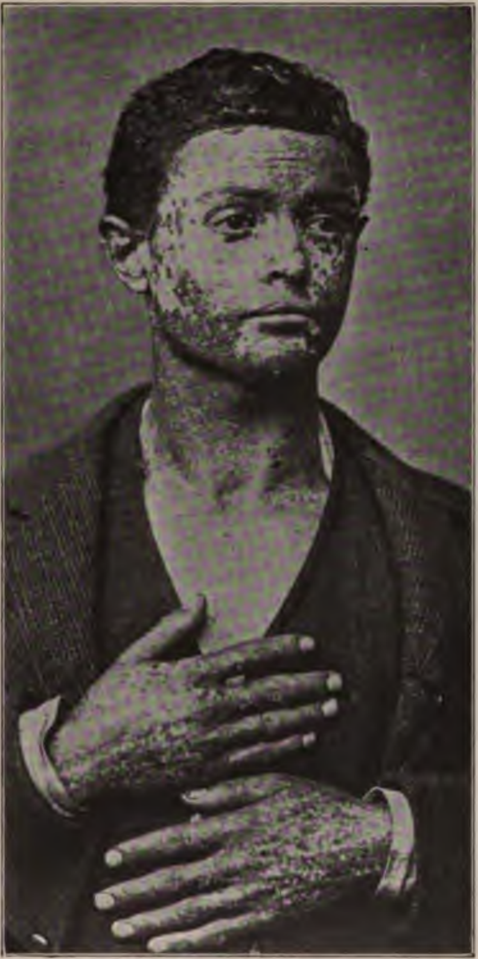

FIG. 252.—XERODERMA PIGMENTOSUM.
(J. C. White's collection.)

many of the cases roderma pigmentosum is a history of attacks thema on the exposed during the early ns of childhood. These sually noticed after ex- e to sunlight. Either or without these ery- tous eruptions, there as the first definite estation of the disease lant freckling; this in- s in intensity so that ately the patients pre- on the exposed parts nost extreme examples e freckled skin. The es are large and prom- and are exaggerated posure to sunlight, and become worse with each ding summer. Ulti- y they give the skin a ed, dark, reddish-brown appearance. With the development of the ing the skin shows atrophic changes similar to those of old age. dry and harsh, and there appear atrophic, white, pitted, punctate and pinhead- to tackhead-size telangiectases. These may develop same time as the freckling, or subsequently.

s time goes on all of these changes become more marked. The skin becomes thin, atrophic, and glistening; keratoses, like senile keratoses, develop; the telangiectases become more prominent, and may become angiomatous. The skin, as a whole, becomes scarlike in appearance, perhaps wrinkled, and shrunken. As a result of these changes increasing the vulnerability of the skin, there are apt to be areas of eczema, and there may develop fissures and ulcers about the orifices of the body, especially the nose, mouth, and eyes. Ectropion and conjunctivitis are characteristic changes; ulcerative keratitis also frequently occurs. In the two groups of cases which I have seen there has been, even before the appearance of keratitis, photophobia; and the position of the head which the patients assume, with head thrown forward and eyes closed until they only see through a narrow slit, has been characteristic.

Sooner or later epitheliomata develop, especially about the face. As they increase there takes place all the hideous changes that come from multiple, large exuberant carcinomata on the face. The eyes are likely to be destroyed by keratitis, and before the end the patients present a shocking picture of destructive disease of the skin, all the more distressing because the victims are usually children.

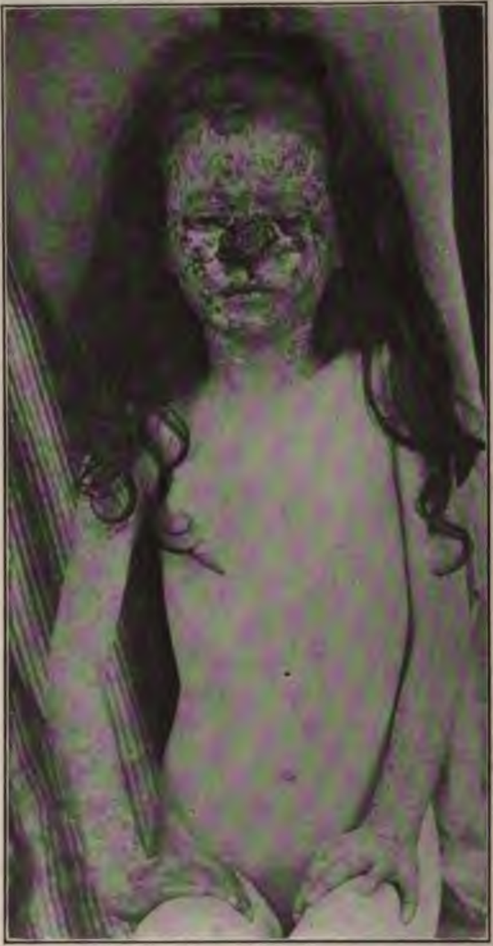
FIG. 252*a*.—XERODERMA PIGMENTOSUM.
(J. C. White's collection.)

The disease shows marked predilection for the exposed parts of the body, and is most marked on the face and backs of the hands. In addition to these areas, its characteristic distribution is the neck, the ioulders, the chest to the level of the third ribs, the forearms, the dorsal irfaces of the feet, and sometimes the legs. Freckling and moderate ianges characteristic of the atrophic skin occur over the body generly, but the extreme developments of the process confine themselves iaracteristically to the areas of predilection. As a rule the disease confined to the skin, but in some cases telangiectases and lentigines e observed on the mucous membranes of the conjunctivæ, lips, and ieeks.

The disease begins typically in early childhood, usually in the first second year of life. It may become manifest within three to five ionths after birth. In some cases tumors begin to appear within a ear after the first manifestations. Ordinarily the progress is less rapid, nd the well-marked degenerative changes and the tumors do not begin ntil five, or six, or seven years of age. Their course then is usually elatively rapid, and in two or three years more the extreme manifestaons of the disease are reached, and the patients in the course of a year r two more die. The progress of the disease may be much less rapid, nd new growths may not begin to appear until the disease has existed r ten or fifteen years, or even longer. In some cases the disease ppears later in life. Occasionally the progress of the disease is not eady, but periods of quiescence occur; and it has been known to ecome stationary before the development of malignant lesions. Kaposi id Hutchinson have recorded cases beginning in young adults, and ises have been recorded by Falcao in which the disease has become ronounced only in old age, in patients who were markedly freckled om infancy.

There are no subjective symptoms in the early stages of the disease. ater the patients suffer the discomforts which result from photophobia nd from such secondary lesions as ulcers and fissures. The epithelimata, as a rule, give the patients little discomfort except when they ecome large. Before the development of malignant lesions the general ealth is unaffected. Late in the course of the disease the patients iffer from cachexia, and ultimately die from marasmus or from deruction of vital structures. Metastases of carcinoma are unusual in ie disease, but have been observed.

Etiology.—The disease is congenital and occurs in family groups. here is no evidence of heredity except, perhaps, in Falcao's case. In early all of the cases reported the disease has shown itself in more ian one child of a family. The sexes are equally susceptible to it, but many of the cases it has confined itself to one sex of the affected mily. In some of the groups of cases it has affected brothers and sters indiscriminately. The typical cases all begin in early childhood, sually in the first or second year, rarely as early as the first three to five months. Exceptionally, it has begun in adult life or old age (Matzenauer, sixty-six; Herxheimer and Hildebrand, seventy years; Falcao, eighty-nine years). It has appeared in the well to do as well as in the poor, and bad hygiene is apparently not a factor in its occurrence. The influence of exposure to light in intensifying the disease has been well marked in many cases, and in Eulenberg's case was the exciting cause.

Unna and Pick have emphasized the influence of light as an exciting cause of the disease, but in Kaposi's opinion this is not borne out by the facts. The consideration of various facts in connection with the disease leaves little room for doubt, in my opinion, that the disease is a congenital defect of the skin which consists very largely in an abnormal susceptibility to light. We have pictures practically identical with xeroderma pigmentosum produced by long-continued exposures to x-rays. I have observed one case strikingly resembling xeroderma pigmentosum in a manufacturer of x-ray apparatus who, for a long time, habitually exposed himself to x-rays. There were changes in the skin of the face, chest, backs of the hands, and forearms which, except for the known cause, would have passed for xeroderma pigmentosum; and epitheliomata ultimately developed in some of the keratoses. In this case we had exposure of normal skin to actinic energy of unusual intensity as the exciting factor of the disease. In xeroderma pigmentosum, as I conceive it, there is the same disturbance of balance between exposure to an irritating agent and resistance that we see in the x-ray atrophy of the skin, but in xeroderma pigmentosum the disturbance of balance is due to an unusual susceptibility of the skin to the ordinarily harmless actinic rays of light. As a result of this extraordinary susceptibility, light produces in these patients the same changes that x-rays produce in normal individuals. Hyde[1] has called attention to the probable influence of light in the production of cutaneous carcinomata. When the ordinary changes of the senile skin have occurred, it is not improbable that the influence of light is an important factor in the production of the pigmentary lesions and of carcinomata. In xeroderma pigmentosum the skin undergoes senile changes of extraordinary degree in a very few years after birth, and responds with extraordinary susceptibility to the influences that produce malignant degeneration in the ordinary senile skin. Among these influences the irritation of the actinic rays of light is probably, in my opinion, the most important.

According to Kaposi, the pathological process in xeroderma pigmentosum begins with proliferation of the connective tissue of the

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[1] *Amer. Jour. Med. Sci.*, January, 1906.

apillæ and with endarteritis, which result in retraction of the papillæ
nd obliteration of some of the capillaries with compensatory dilata-
on of others. There is irregular accumulation of pigment in the rete,
owngrowth of the rete pegs into the corium, and proliferation of the
utaneous glands and degeneration of their epithelium. Crocker found
ome of the tumors to be distinctly papillomatous, and not malignant.
'he tumors are, as a rule, carcinomata, frequently melanotic. Kaposi
nd others have described them as carcinomata and sarcomata, but
elanotic carcinomata have probably been mistaken for sarcomata.

Diagnosis.—The occurrence of the disease in more than one mem-
er of a family, and the striking character of its symptoms leave almost
o room for confusion in diagnosis. In the early stages of the disease,
efore the development of serious lesions, it would be strongly suspected
rom the extreme degree of freckling and the premature development
f atrophic senile changes in the skin. There is no other disease which
losely resembles it.

Prognosis.—The cases are hopeless. After the development of ma-
gnant growths, they die within a few years. Malignant growths usually
evelop within ten to twelve years after the first manifestations of the
isease, but have been delayed from ten to thirty years. Nothing is
nown which influences radically the course of the disease. Probably
crupulous protection from exposure to light would be the most effectual
recaution which could be taken against its development. This would
ecessitate the wearing of clothing, including a covering for the face,
which is entirely impervious to all light rays except red; but the dis-
ase is so hopeless that the precaution would be warranted in children
howing the first manifestations of the disease who had brothers and
isters in whom it had already developed. The local treatment is symp-
omatic, and consists in the use of boric-acid solution or other lotions
or the eyes and in the treatment of ulcers and other lesions in the usual
ways. In exceptional circumstances it may be advisable to excise or
therwise destroy the epitheliomata.

III.—ATROPHODERMA ALBIDUM[1]

SYNONYM: *Pityriasis alba atrophicans* (*Krösing*).

Kaposi describes a "second form of xeroderma" which is a station-
ry condition of atrophy of the skin. The epidermis is thin, glistening,
nd wrinkled. It is poor in pigment, unusually white, and in places
tense and not easily lifted. It affects the skin of the legs from the

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[1] Kaposi, p. 501.—Jadassohn, *Fourth Germ. Derm. Congr. Trans.*—Krösing,
rm. Zeitschr., vol. iii (1896), p. 57.

sole to the middle of the thigh, and occasionally that of the arms and hands, including the palms. On account of the thin epidermis, the tips of the fingers and the palms and the soles are tender and more vulnerable than ordinary. The condition remains stationary from early childhood, and is apparently a congenital defect of the skin analogous to nævus, from which, however, it is distinguished by the distinct thinning of the skin. Similar cases have been described by Krösing and by Jadassohn.

Its treatment consists of bland ointments to lubricate the skin.

IV.—DIFFUSE IDIOPATHIC ATROPHY OF THE SKIN[1]

Synonym: *Dermatitis atrophicans* (Kaposi).

Rare cases have been reported from time to time in which diffuse atrophy of the skin has occurred without apparent cause. Some of these cases are anomalous forms of xeroderma, and possibly of other dermatoses. In others there is a true idiopathic atrophy which cannot be regarded as the sequel of any other cutaneous disease.

Symptomatology.—The only constant characteristic of the cases is simple atrophy of the skin without inflammatory symptoms and in many cases without the manifestations of any other disturbance. The skin becomes thin; in some cases its surface is crackled or cigarette-paperlike; in others it is smooth and shiny and parchmentlike. There may be looseness and wrinkling of the skin. The atrophy may not be equal in the involved areas, but is in some cases mottled, showing as numerous coin-sized patches of atrophy around which the skin is less atrophic. There is usually atrophy of the appendages, with partial or complete loss of hair and diminution of sweat and sebaceous matter. The skin becomes dry, and there is at times branny desquamation.

There may be evidence of vascular disturbances in the diseased areas, with dilatation of the superficial capillaries and at times the formation of reddish or purplish areas. There may be also disturbances of pigment, with the occurrence of pigmented spots and patches. In pronounced cases, ulcers are apt to form over bony prominences.

The condition is usually acquired and develops slowly, but in rare cases the development has been rapid. It is as a rule partial, and is ost frequent upon the legs. In a few rare cases it has become almost quite universal. In most cases it has been symmetrical, but it has en observed to be unilateral. Objective symptoms have usually been king. A few cases have had shooting pains, or shown tenderness the atrophic areas.

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[1] Schwimmer, "Die neuropathischen Dermatosen," p. 189.—Atkinson, *Richmond and Louisville Med. Jour.*, vol. xxiv, 1877.—Buchwald, *Archiv*, 1883, p. 553.—Touton, *Deutsch. med. Wochenschr.*, 1886, p. 6.—Pospelow (2 cases), *Annales*, 1886, p. 505.—Williams, *Brit. Jour. Derm.*, 1894, p. 342.—Bronson, *Jour. Cutan. Dis.*, 1895, p. 1.—Elliot, *Jour. Cutan. Dis.*, 1895, p. 152.—Fordyce, *Jour. Cutan. Dis.*, 1897, p. 230.—Kaposi, *Archiv*, 1897, vol. xxxix.—Neumann, *Archiv*, 1898, vol. xliv, p. 3.—Colombini, *Monatshefte*, 1898, vol. xxviii, p. 65.—Holder, *Jour. Cutan. Dis.*, 1899, p. 37.

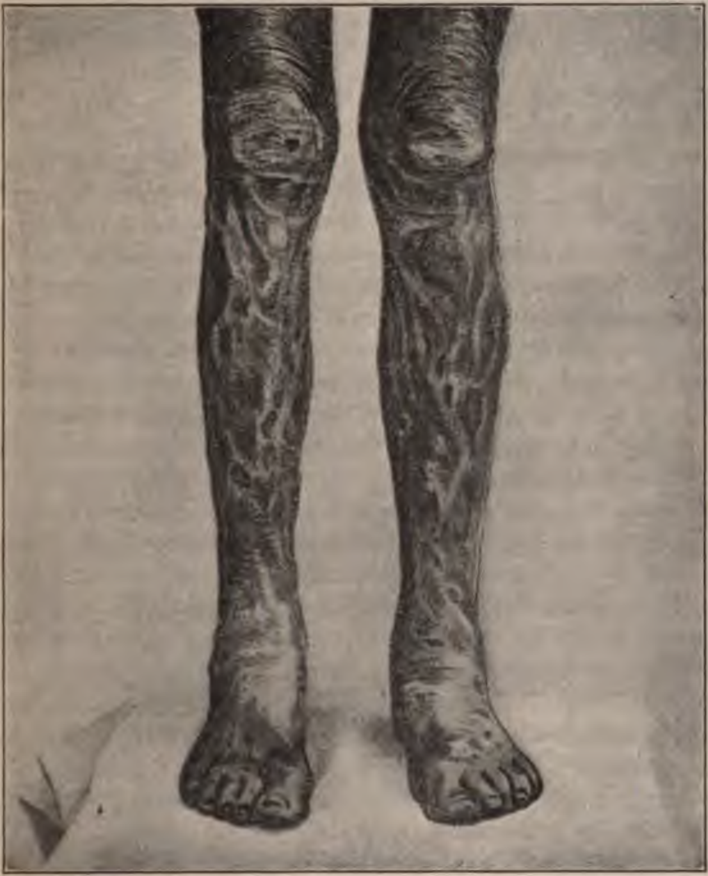

FIG. 253.—SYMMETRICAL ATROPHY OF THE SKIN.
(Photograph of Bronson's plate.)

Etiology and Pathology.—The etiology of the cases is complex. any of them have appeared in patients apparently healthy without y assignable cause. Previous chilling of the body has been assigned the cause in some of the cases. In a few cases the condition has en congenital. In most cases it has appeared in adult life or old age.

In some of the cases the atrophy has been secondary to a slight flammatory process. In others it is primarily a trophic process resulting in atrophy. In some of the cases vascular changes have been observed analogous to those of morphea, and it is probable that in many of the cases the condition is closely related to morphea. Some of the cases have been syphilitic.

Prognosis and Treatment.—The course of the affection is usually slowly progressive, but in cases observed by Kaposi and Colombini the atrophy became almost universal in five or six months.

Treatment is unavailing.

V.—KRAUROSIS[1]

SYNONYM: *Kraurosis vulvæ.*

Kraurosis is a term applied to an atrophy of the skin of the external genitals in women. It was first described by Breisky in 1885.

Symptomatology.—The external genitals, especially the labia minora, preputium clitoridis, and the clitoris, become shriveled and atrophied. The labia minora and the clitoris may entirely disappear, their sites being replaced by two furrows and a minute depression. The skin of the involved parts is shrunken, and the shrinkage may be sufficient to cause constriction of the vaginal orifice. There are frequently gray, roughened plaques of leukoplakia. The surrounding skin is usually dry and shiny, and whitish or of a reddish-gray color. Epitheliomata may develop late in the course of the affection. The appendages of the skin share in the atrophy, and there is thinning or loss of the hair. Its early course is usually associated with pruritus which, however, usually disappears in the later stages of the disease.

It is a disease of adult or later life. Some of the cases have developed without preceding disturbances, but in many cases the condition has been preceded, and its early course accompanied, by severe pruritus. In other cases it has followed eczema and other inflammatory disturbances. An association with vaginal discharges is not uncommon, but there is apparently no relation to sexual habits.

Anatomically, the condition would seem to be primarily an inflammatory process, resulting in sclerosis and atrophy of the corium and destruction of the appendages of the skin.

Prognosis and Treatment.—The condition, when once established, is, like other atrophies, permanent. Pruritus, eczema, and vaginal discharges are treated in the usual ways. Patches of leukoplakia should be treated either by destruction or the application of x-rays, and total removal of the parts may be justified where the leukoplakia is abundant and the danger of epitheliomata imminent.

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[1] Breisky, *Zeitschr. für Heilkunde*, 1885, p. 69.—C. Heitzmann, *Trans. Amer. Derm. Assoc.*, 1888.—Ohmann-Dumesnil, *Monatshefte*, 1890, vol. x, p. 294 (review and bibliography).—Baldy and Williams, *Amer. Jour. Med. Sci.*, vol. cxxviii, 1899, p. 528 (bibliography).

VI.—STRIÆ ET MACULÆ ATROPHICÆ[1]

Synonyms: *Lineæ albicantes, Atrophoderma striatum et maculatum.*

These are the glistening, whitish lines and spots which are seen on the skin usually as a result of stretching, as in pregnancy.

The lineæ albicantes which occur upon the abdomen after pregnancy are typical examples of the condition. In the linear form of the affection there occur streaks which appear as though the skin had been ruptured beneath the epidermis. They are pearly or bluish white, thin and glistening, and level with the skin or slightly depressed. They are usually not more than a quarter of an inch wide, and vary from one o several inches in length, their direction following the lines of cleavage of the skin. At the time of the formation of these streaks there is usually hæmorrhage into the areas, or inflammatory symptoms which disappear slowly, leaving the permanent, scarlike lines.

The atrophic spots are similar in character to the streaks, but occur ess commonly. They vary in size from spots a quarter of an inch or ess in diameter to patches the size of a coin. They may be discovered n the beginning as hæmorrhagic or red patches, which are succeeded by the bluish-white, apparently atrophic patches. Both the streaks and he patches may diminish in size after their development from the ontractility of the skin, but they are permanent and do not entirely disappear.

The striæ are usually symmetrical in distribution. They occur most frequently over the lower part of the abdomen, the trochanters and xternal surfaces of the thighs, the buttocks, and the anterior borders of the ilia; but either the streaks or the patches may occur anywhere, s result of the distention of the skin or from other mechanical causes. They are without objective symptoms, except, perhaps, tenderness at he time of their development.

The atrophic striæ are very common lesions. The maculæ are much ess common. Schultze found the striæ or maculæ in thirty-six per ent of women who had never borne children and in six per cent of men. The striæ are very common in fat men, and, independent of pregnancy, their greater frequency in women is probably due to the usual presence of a larger amount of adipose tissue. The lesions may occur n childhood, but they are unusual before early or middle adult life.

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[1] Shepherd, *Jour. Cutan. Dis.*, 1891, p. 59 (bibliography).—Sherwell, *Jour. Cut. nd Gen.-Urin. Dis.*, vol. xii, 1894, p. 499.—Bunch, *Brit. Jour. Derm.*, January, 1905.

The lesions in nearly all cases are probably due to mechanical causes, usually to rapid distention of the skin, as from pregnancy, anasarca, the rapid accumulation of fat, or development of tumorlike lipomata. Occasionally they are observed to follow serious illness. In these cases their presence may be due to the rapid accumulation of fat during convalescence, but in some of the cases they are probably the result of nutritional disturbances in the skin itself. They may result from external traumatism, as from a blow, which may not cause a break in the epidermis.

Anatomically, Kaposi found separation of the connective-tissue bundles of the corium, obliteration of the papillæ, diminution in the number of vessels, glands, and adipose globules in the skin, and atrophy of the epidermis.

The lines can hardly be confused with any other lesion. The macules may be mistaken for morphea; they are, however, thin, pearly or bluish white and atrophic, without the ivorylike appearance, the density, or the lilac border of morphea.

The lesions are permanent and cannot be removed by treatment.

VII.—MULTIPLE, BENIGN, TUMORLIKE, NEW GROWTHS

Under this name Schweninger and Buzzi[1] describe a case in which small, soft, tumorlike lesions occur, due to a kind of ballooning of atrophic spots of the skin. The condition is excessively rare, but cases have also been observed by Morris, Colcott Fox, and Van Hoorn, and Stelwagon and Crocker each refer to cases in their experience. The lesions are whitish, round or oval, slightly elevated swellings, from one half to two centimeters in diameter, and flatten under the touch. Their sites of predilection are the shoulders and back and the thighs, and the lesions are usually numerous. They may undergo spontaneous involution, leaving soft, depressed scars, or they may shrink without disappearing; there is, however, a tendency for new lesions to appear, thus gradually increasing the extent of the disease. They cause no discomfort.

Their etiology is unknown. Four of the five cases have been in women. It is probable, as Crocker states, that the lesions are the result of more than one pathological process.

Buzzi found in all of the lesions examined absence of the elastic fibers in the involved skin, with slight increase around the borders, and this passive retraction of the elastic tissue was apparently the primary pathological change.

They are uninfluenced by treatment.

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[1] "International Atlas of Rare Skin Diseases," Part V.

VIII.—GLOSSY SKIN[1]

SYNONYM: *Atrophoderma neuriticum.*

Glossy skin is an atrophy of the skin following injury or disease of he nerve supplying the affected area.

The condition was first described by Paget, and Mitchell, Morehouse nd Keen in 1864, as a result of local nerve injuries.

Symptomatology.—The disease involves the extremities, particurly the hands, and is characterized by a condition of the skin which glossy" well describes. The skin becomes smooth, shiny, and scarlike. he affected fingers become tapering, smooth, and waxlike; they may be ink or red, or mottled pink and white. When the mottling is proounced the condition may resemble very closely chilblains. The apendages of the skin are involved so that the skin is dry and hairless, lthough occasionally there is increased sweating on the palms and the ondition may be accompanied by palmar and plantar keratoses. The ails show nutritional changes; they are curved longitudinally and transersely, and there is often thickening of the epidermis under the free order. The retraction of the atrophic skin on the bulbs of the fingers ay expose the sensitive matrix and result in the formation of fissures nd ulcers which may be very painful, particularly upon the toes. The ondition occurs on the extremities and may involve the entire distal ortion of the extremity, or may be confined to one or two digits. Acompanying and perhaps preceding the objective symptoms of the disase, there is usually severe and persistent burning pain (causalgia) hich is characteristic.

Etiology and Pathology.—The affection is a trophic disturbance econdary to disease of the nerve supplying the affected area, and may e produced either by injury or disease. It most frequently occurs as esult of injuries, but is occasionally seen in the course of various diseases ccompanied by neuritis, such as gout, rheumatism, chronic myelitis, nd leprosy.

The disease may be closely simulated by conditions of feeble periphral circulation. Hyde and Montgomery, indeed, suggest that some f the cases of glossy skin are the result of circulatory disturbances fter exposure to severe cold, and call attention to the relation of glossy kin to other disturbances of peripheral circulation, like Raynaud's isease.

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[1] Paget, *Med. Times and Gazette*, 1864, vol. i, p. 333.—Mitchell, Morehouse nd Keen, "Gunshot Wounds and Other Injuries of the Nerves," Philadelphia, 1864, . 77.—Mitchell, "Injuries of Nerves and their Consequences," Philadelphia. 1872.—eloir, "Twentieth Century Practice," vol. v, p. 834.

Diagnosis.—The development of the peculiar, shiny skin upon the extremities, in association with neuritis, is characteristic. Raynaud's disease, which it most closely resembles, is symmetrical and is not associated with well-defined neuritis.

Prognosis and Treatment.—The affection ultimately disappears, but it may continue from a few weeks to several years. The general treatment is directed to the underlying disease. Local treatment consists in the use of soothing applications and protection especially from exposure to cold. Immersion in very hot water is useful for relieving the causalgia.

IX.—PERFORATING ULCER OF THE FOOT[1]

SYNONYM: *Malum perforans pedis.*

This term is applied to a deep, round ulcer or sinus which develops on the under surface of the foot, rarely upon the hand, in association with degenerative nervous disease.

Symptomatology.—The lesion usually begins as a mass of thickened epidermis, like a corn under which ulceration takes place. From the thickening of the surrounding epidermis a horny, hypertrophic border is formed, in the center of which there is found a sinus, or a round ulcer having a wall of unhealthy granulations. The ulcer is usually very deep, involving the bone beneath, and not infrequently causes disorganization of the metatarso-phalangeal joint. There is usually very little discharge. The ulcer usually develops at the point of greatest pressure, typically on the ball of the foot, behind the great toe. Occasionally it occurs on the heel, or beneath the other metatarso-phalangeal joints. The lesions have been seen upon the hand and wrist. There is usually one lesion, but there may be more than one. The ulcer itself is, as a rule, quite insensitive, although walking may be very painful. Occasionally the ulcer is hypersensitive. As concomitant symptoms develop there may be anæsthesia, neuralgic or rheumatic pains, and coldness of the part. The condition is frequently associated with other evidences of nutritional disturbances, like alteration of the nails, anidrosis, hyperadrosis, hyperpigmentation, and diffuse hyperkeratosis.

Etiology and Pathology.—The affection is essentially a trophic disturbance, but is excited by mechanical causes. It may be produced either by damage to the nerve center, as in tabes, to the nerve trunk, as in leprosy or syphilis, or to the nerve terminals, as in peripheral neuritis. It is most frequently seen in adults and in a vast majority of cases in males. In Gasguel's statistics, of 91 cases, 84 were males. Three were ler twenty; 4 between twenty and thirty; 22 between thirty and forty; between forty and fifty; 19 over fifty. In 69 there were central nerve ons; 32 had tabes; 17, general paralysis; 12, various cord lesions; 4, ımatic disease of the cord; 8, various cord lesions; 1, Friedreich's xia. In 8 there were peripheral nerve lesions; in 14 there was diabetes.

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[1] Savory and Butlin, *London Medico-Chirurg. Soc. Trans.*, vol. lxii, 1879, p. 373.—Gasguel, *Thèse de Paris*, July, 1890.

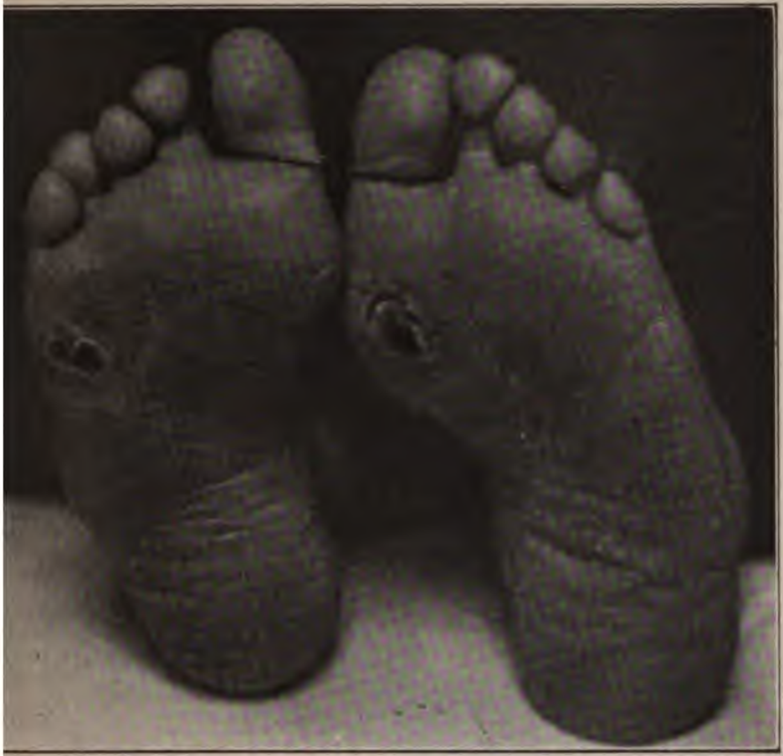

Fig. 254.—Perforating Ulcer. (Author's collection.)

Diagnosis.—From a simple, suppurating corn, and from other erative lesions of the foot, it is readily distinguished by its peculiar nd shape, by its painless and persistent character, and by the presence other evidences of degenerative nervous disease.

Prognosis and Treatment.—The lesion is usually permanent. It y be healed by entire disuse of the foot, but recurs upon renewed empts to walk. The treatment of the lesion itself is by curettement or er surgical measures, including amputation. But all surgical pro- ures are liable to failure; the ulcer may even appear in the stump er amputation.

X.—AINHUM[1]

Synonyms: *Ainhun, Dactylolysis spontanea.*

Etymology: Said to be from an African word meaning "to saw." Matas derives it from the word ainhoum, of the negro patois of Brazil, meaning "fissure."

Symptomatology.—Ainhum is a disease characterized by the formation of a constricting ring around one or more of the digits, usually the little toe, and the gradual spontaneous amputation of the affected part.

It was first described by Clark in 1860; later attention was attracted to it by Da Silva Lima in 1880.

The disease is local, and begins with the formation of a crescentic transverse furrow usually in or near the digito-plantar fold of the little toe. This furrow begins without inflammatory or subjective symptoms and without any breach of the surface. The furrow gradually increases in depth and length, and finally produces a constricting ring around the toe. From interference with the circulation of the part the toe beyond the constriction is usually swollen to two or three times its normal size, and as time goes on and the constriction becomes more marked the toe looks like a tumor attached by a pedicle. Before the constriction becomes marked there may

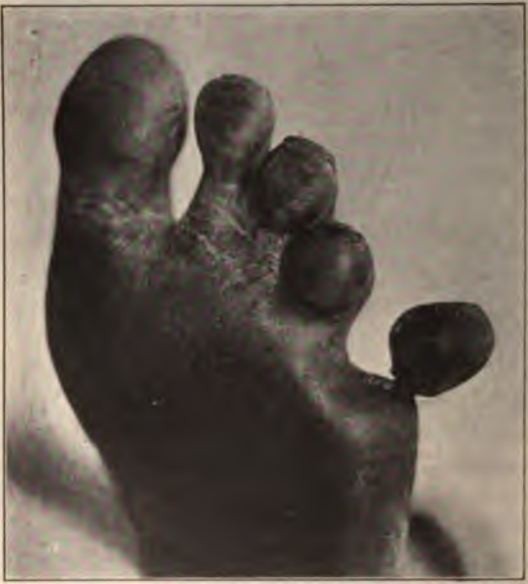

Fig. 255.—Ainhum. (Herrick's photograph.)

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[1] Clark, *Trans. Epidemiolog. Soc.*, 1860, vol. i, p. 105.—Da Silva Lima, *Arch. Derm.*, 1880, p. 367.—Duhring, *Amer. Jour. Med. Sci.*, vol. lxxxvii, 1884, p. 150.—Duhring and Wile, *Amer. Jour. Med. Sci.*, January, 1884.—Eyles, *Lancet*, 1886, ii, p. 576 (histology).—Shepherd, *Amer. Jour. Med. Sci.*, vol. xciii, 1887, p. 137.—Pyle, *Medical News*, 1895, i, p. 85 (complete bibliography).—Ohmann-Dumesnil, "Medicine," 1895, p. 202 (bibliography).—Matas, "Surgical Peculiarities of the Negro," *Trans. Amer. Surg. Assoc.*, vol. xiv, 1896.—Herrick, *Phila. Med. Jour.*, 1898, i, p. 246.—Manson, "Tropical Diseases" (full bibliography).

no swelling. As the constriction becomes very narrow the distal rtion of the toe is twisted outward, and ultimately spontaneous am- tation occurs. This may occur without ulceration, or it may be pre- ded by an ulcerating fissure with fetid discharge. The amputation ay occur at the first, second, or third joint of the digit, or less fre- ently through the phalanx.

The affection typically involves the little toe, but it may involve any the toes, and in some cases involves both toes and fingers. The ring usually near the proximal joint of the digit, but may occur anywhere in length.

The course of the disease is very slow, requiring as a rule from four ten years for amputation, or even much longer—fifty years (Evans). e condition may be associated with vasomotor and trophic changes in e affected limb, and perhaps with sensory disturbances. The condi- n is unaccompanied by characteristic subjective symptoms. Toward e end the toe may be a source of trouble from its liability to mechan- l injury, and when ulcers occur they may be painful.

Etiology and Pathology.—Ainhum is a disease of the dark races, rticularly of negroes, but has been observed in white subjects. It is en chiefly in Africa and India, and among the negroes of South Amer- a and the West Indies. A few cases have been recorded in America. errick has reported a case in a negro who for thirty years had lived in linois. It is most frequently seen in men, and occurs usually in young lults, rarely in old age or childhood. In a case reported by Duhring, d in some other cases, there has been a history of the affection in pre- ious generations, although heredity seems a doubtful factor.

Further than this its etiology is unknown. Its occurrence in negroes apparently a manifestation of their well-known susceptibility to fibrous- ssue formations, and one explanation of its occurrence is that it is a sult of injury of the exposed parts. It seems more likely that the affec- on is a trophic disturbance, perhaps analogous to or identical with cir- mscribed scleroderma—an annular scleroderma. Manson has observed case accompanied by scleroderma of other parts, and Stelwagon has bserved a case involving both fingers and toes, in association with a ndition suggestive of pityriasis rubra pilaris. Such cases are impos- ble to explain upon the theory of mechanical injury, and strongly sug- est the trophoneurotic character of the affection. It is possible that inhum is caused by the presence of filaria perstans. This organism found especially upon the west coast of Africa, and according to a cent report of Wellman (*Journal American Medical Association*, Janu- ry 19, 1907), its intermediary host is a tick through whose bite it is ansferred from man to man.

Anatomically there is a ring of fibrous tissue covered by a thickened, horny epidermis. When the constriction affects the bone there occurs a rarefying osteitis. According to Eyles, the primary change is in the epidermis, and consists of increase of the horny layer and downgrowths of the interpapillary pegs of the rete.

Diagnosis.—Ainhum is distinguished from mutilating leprosy, syringomyelia, Raynaud's disease, and various ulcerative processes occurring from nervous disease by the fact that it is not accompanied by any of the other symptoms which characterize these affections. It may be remembered, however, that amputations may occur in leprosy, syringomyelia, and Raynaud's disease which resemble more or less closely those following ainhum.

Treatment.—Da Silva Lima and others have found that the progress of the ring may be checked in its early course by transverse incision. The only other treatment is amputation when the part becomes troublesome.

XI.—SYRINGOMYELIA[1]

SYNONYMS: *Morvan's Disease, Analgesic Paralysis with Whitlow.*

Syringomyelia is a disease of the spinal cord producing sensory disturbances and trophic changes which are manifested chiefly in the upper extremities.

Symptomatology.—The first evidence of the disease is usually pain in one or both arms, accompanied by loss of muscular power. These symptoms are followed by analgesia; occasionally the analgesia is the first symptom noticed. Following later there is typically a succession of whitlows. These may be multiple at one time, but are usually single. They may rapidly succeed one another, or they may be separated by intervals of several years. They usually occur on the fingers, occasionally on the toes, and they are followed by necrosis and loss of the phalanges. Other trophic and vasomotor changes also appear. There is coldness and cyanosis of the extremities, with a tendency to the formation of bullæ and with nutritional changes in the nails. Glossy skin may develop, and ulcerations occur which may extend into the sheaths of the tendons. There is atrophy and paralysis of the muscles of the hands and of the flexor muscles of the wrists, and the development of claw-shaped hands. Scoliosis and arthropathies are common. The sense of touch is not affected, but there is loss of the involved areas of heat, cold, d pain sensibility. The affection usually involves first one and then e other upper extremity, but it may be confined to one. It may also ack the legs. In rare instances it may attack one arm and one leg.

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[1] Morvan, *Gazette hebdom.*, 1883, pp. 580, 590, 624, and 721.—Sachs and Armstrong, *New York Med. Jour.*, 1892, vol. lv, p. 482 (bibliography).—Dyer, "Syringomyelia and Lepra nervorum," *New Orleans Med. and Surg. Jour.*, vol. xxi, 1893-94, p. 81.—Cagney, "Syringomyelia and Leprosy," *Brit. Jour. Derm.*, 1894, p. 375.—Schlesinger, "Die Syringomyelie," Leipzig and Vienna, 1895 (complete review and bibliography).

Its etiology and pathology are obscure. For their consideration ref- ence must be made to works on neurology.

Diagnosis.—The clinical resemblance to anæsthetic leprosy may be ry close. In the very rare cases of leprosy which are not sufficiently stinct to make the diagnosis positive, it can readily be established by e finding of the leprous bacilli in the tissues. The nervous symptoms syringomyelia would readily distinguish it from any pure dermatosis.

Prognosis and Treatment.—The disease is progressive and treat- ent is palliative. The constitutional treatment of the condition is purely mptomatic and consists in supporting measures. The local treatment nsists in the protection of the parts from injury, and the treatment of e ulcers, whitlows, and other cutaneous lesions that occur according to dinary methods.

SECTION XI

ANOMALIES OF PIGMENTATION[1]

THE anomalies of pigmentation form a small but interesting, and, from the standpoint of pathology, obscure class of affections. These anomalies may consist of excess or deficiency of pigment, and they may be either acquired or congenital.

A. INCREASED PIGMENTATION

Increased pigmentation of the skin may be due either to the increase of normal pigment (melanin) or to the deposit of blood pigment (hæmosiderin) in the skin. Because of their clinical resemblances these two forms of hyperpigmentation are considered together, although they are, of course, entirely distinct pathological processes. Pigmentation due to deposit of blood pigment in the skin follows pathological processes in which red blood corpuscles are extravasated in the skin and there undergo decomposition with deposit of their pigment in the tissues. Such pigmentation is but one step in the involution of hæmorrhage into the skin, which has been considered under Purpura.

True hyperpigmentation (melanosis) is produced by the presence of an increased quantity of melanin in the lower part of the mucous layer and in the papillary layer. The quantity of pigment is greatest in the basal layer and in the tissues closely adjacent thereto, but it occurs in gradually diminishing quantity upward through the mucous layer and downward in the papillary layer. The pathology of the production of hyperpigmentation is various. The stimulation of the cells which is produced by an ordinary inflammatory process in the skin is frequently followed by the occurrence of hyperpigmentation. We see this after artificial dermatitis, as from the application of a chemical irritant, or after a burn of the first degree, or in areas continually chafed by clothing, in old cases of body lice where the skin has been constantly inflamed by scratching, and in various other inflammatory conditions. Hyperpigmentation associated with inflammation, but probably, at least in part, e to the direct effect of actinic energy upon the pigment-forming tis- es, is seen from the effect of sunlight and x-rays. Hyperpigmentation so occurs as the result of long-continued use of drugs, notably of senic. Here the process seems to be analogous to that of its produc- n by external irritation, for arsenic has a selective irritative effect on the cells of the skin.

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[1] von Düring, *Monats.*, vol. xlii, No. 10, Abs. (elaborate paper).

Melanosis occurs in certain general diseases, as Addison's disease, abetes, and various diseases of the liver. Hyperpigmentation in cir- mscribed areas is seen occasionally in the secondary period of syph- s; frequently during pregnancy and in association with uterine orders. The frequent association of hyperpigmentation with preg- ncy, carcinoma of the abdominal viscera, and other abdominal and lvic disturbances suggests strongly that irritation of the abdominal mpathetic, especially of the solar plexus, is the causal factor, but of pathological mechanism which produces hyperpigmentation in such ses we are totally in ignorance.

Among skin diseases hyperpigmentation is curiously associated with ultiple fibroma of v. Recklinghausen, and it is a sequence of many flammatory diseases of the skin like lichen planus, psoriasis, and der- atitis herpetiformis.

The following attempt at a complete classification of the cutaneous perpigmentations is adapted from Unna's classification, and is inserted order to give a summary of all the forms:

) Melanosis, due to excessive deposit of true pigment:

(*a*) Chemical; following the stimulation produced by chemical ritants.

(*b*) Actinic; produced by actinic agents (sunlight, x-rays, heat); eckles, tanning.

(*c*) Toxic; pigmentary syphilide, Addison's disease, arsenical pig- entation.

(*d*) Reflex, or nervous; examples of which are the freckling and tches of pigmentation seen in pregnancy and various disturbances of e abdominal and pelvic viscera. Acanthosis nigricans.

2) Hæmosiderosis, due to deposit of blood pigment in the skin:

(*a*) Post-hæmorrhagic pigmentation.
(*b*) Various chronic stagnation pigmentations.
(*c*) Sarcomatous pigmentations.
(*d*) Ulceration and scar pigmentations.

Discolorations due to the deposit of extraneous pigment in the skin e also to be considered here; among these chiefly to be mentioned are ttoo marks and argyria.

I.—LENTIGO

SYNONYMS: *Freckles, Ephilides.*

Characteristics.—Lentigines, or freckles, are a familiar condition. They occur as yellowish or brownish-red spots, without elevation, roundish or somewhat irregular in shape, and from the size ordinarily of a pinhead to a tackhead, occasionally considerably larger. In color, as a rule, they vary from a faint yellow to a brown. Extreme lesions may be dark brown or even black, or occasionally greenish in color. Freckles may be limited to a few spots upon the face and hands. Ordinarily, when they occur, they are more or less abundant. As a rule, they occur chiefly on the exposed parts of the body. They are especially common on the face, particularly over the bridge of the nose and on the cheeks. They also occur commonly on the backs of the hands and over the back and front of the upper third of the chest. In extreme examples of freckling it is not uncommon to find faint freckles generalized over the entire body.

The distribution of freckles is usually quite symmetrical. Rare cases of distinct unilateral freckling have been recorded; in a few cases the freckling has followed the course of one nerve, as the supraorbital, or the second division of the fifth. Occasionally the development of freckles is confined to unusual localities. I have observed a woman, aged forty, in whom during the last nine years there has been the slow development of an abundant crop of freckles upon the inner and outer aspect of the thighs and upon the back, accompanied by a very few faint freckles upon the face. There is no pathological condition to account for the occurrence, although during this period she has had one pregnancy and an operation for shortening the round ligaments, to which she attributes the freckling. Crocker has recorded a similar case in a woman, aged twenty-six, who was anæmic and constipated, and "held a position of anxious responsibility." Occasionally freckling occurs upon the mucous membranes of the lips and mouth, as has been observed by Hutchinson and Balzer. I have observed freckling on the mucous border of the inner surface of the lips of a woman, aged forty, of highly nervous temperament, who was under treatment for facial eczema. The patches were roughly rectangular, about a quarter of an inch in long diameter, and very dark brown. They appeared some months after the first attack of eczema, apparently independently of the eczema, and continued for many months after the eczema ceased. In the course of a year, when her nervous condition improved, they entirely disappeared. Duhring has observed cases in which the freckling was confined to the buttocks and penis.

Freckles, when once developed, are usually permanent. They become more marked after exposure to light or other pigment-exciting factors, t even in the absence of these, they usually remain faintly apparent. is not uncommon, however, for freckles to be very abundant on the ce in childhood and in later life almost completely to disappear. Frec- es also which are produced by pathological disturbances tend to disap- ar upon the removal of the cause.

Etiology and Pathology.—Freckles are in many cases a congenital fect, although they do not, as a rule, appear until pigmentation of the in begins to be marked, so that they are rarely seen before four years age. They may, however, be apparent at birth. They are usually st apparent during the second decade of life. Most extreme freckling seen in persons of red or sandy hair, and, like that peculiarity, ckles are often hereditary. Freckles are exaggerated by exposure to nlight and also, under exceptional circumstances, by exposure to other rms of actinic energy, as heat or x-rays.

Freckling, or patches of pigmentation, which are yet to be considered der the head of chloasma, are occasionally symptomatic of other con- tions. The appearance of large freckles is common in the senile skin, d is frequently the first stage of senile keratosis. Extreme freckling also the first stage of xeroderma pigmentosum. The appearance of eckles is occasionally seen in abdominal or visceral carcinoma.

Anatomically freckles are circumscribed patches containing an in- eased amount of pigment in the rete.

Prognosis and Treatment.—Freckles can usually be temporarily re- oved, but their prompt recurrence cannot be prevented except by care- l avoidance of exposure to exciting causes, which is usually too great price to pay for relief.

The principle of the methods for their removal is to cause desqua- ation of the epidermis, and thus remove the rete cells containing the gment. For this purpose bichloride of mercury is the most efficient ent, in the strength of 1 to 200, or 1 to 500:

℞ Mercuric chloride	gr. i–ij;
Zinc sulphate	gr. v–x;
Tincture of benzoin, Alcohol, Water, āā	ʒiij.

his should be dabbed on the surface three or four times daily until des- amation is produced.

Individual lesions may be carefully painted to produce active desqua- ation with:

Lactic acid	1	part;
Water	5–20	parts.

The lesions may be treated more vigorously by the application of a paste or plaster containing five to twenty per cent of salicylic acid alone or combined with an equal quantity of resorcin. Such an application is applied until it produces active inflammation and desquamation. These vigorous applications are only to be used where the patient can give herself up to the treatment for several days, and are only advisable where the demand for relief is insistent. Ordinarily the use of a bichloride and alcohol solution is sufficient.

II.—CHLOASMA

Synonyms: *Moth Patches, Liver Spots.*

The term chloasma is applied to patches of hyperpigmentation of the skin. Chloasma is divided into two varieties, idiopathic chloasma and symptomatic chloasma.

Idiopathic Chloasma is the term applied to the various forms of hyperpigmentation due to external agencies, such as exposure to sunlight, x-rays, and irritating drugs. These forms require no detailed description. Under this variety are included:

Chloasma caloricum, from exposure to heat.

Chloasma toxicum, from the application of irritating drugs.

Chloasma traumaticum, from mechanical irritation, as from the friction of a garter or the irritation produced by scratching.

Symptomatic Chloasma is the term applied to the patches of pigmentation which accompany various systemic disturbances. The condition is seen with greater or less frequency in secondary syphilis, tuberculosis, malaria, cancer, Addison's disease, utero-ovarian disturbances, and pregnancy. As a matter of fact, while the definition of chloasma includes all of these forms of pigmentation, the so-called idiopathic chloasma and the pigmented areas which occur symptomatically in Addison's disease and occasionally in other diseases are not usually spoken of as chloasma, but simply as increased pigmentation. When the term chloasma is used, it nearly always refers to the patches of pigmentation which occur chiefly about the face, and which are not, as a rule, associated with any well-defined systemic disease.

Symptomatology.—The sole symptom of chloasma is the occurrence of increased pigmentation in the patches. There are no subjective symptoms, and there are, as a rule, no other changes in the skin, although occasionally there is oily seborrhœa of the patches. The patches are of chamois, or slightly darker, brown color. They are of irregular or roundish shape, sometimes with sharp borders, sometimes with borders merging gradually into the surrounding skin. The patches are usually few, occasionally there are several which may coalesce into irregular eas. They are found upon the face, especially the forehead, less equently on the trunk and other parts. As a rule, they are without mmetry in their distribu- on. Their development is dinarily slow, but they may pear quickly. The same is ue of their disappearance. loasma uterinum, which oc- rs in pregnancy as well as in thological conditions of the erus and ovaries, appears iefly on the face. It may o appear on the breasts, ab- men, and occasionally on her parts.

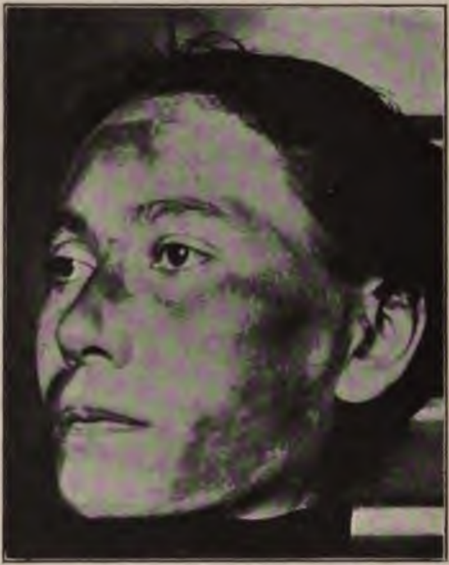

FIG. 256.—CHLOASMA, UNUSUALLY DISTINCT. (Heidingsfeld's collection.)

The hyperpigmentations ich are symptomatic of vari- s constitutional diseases are t limited to a few patches, ch as are described above, t have a generalized dis- ibution which is more or ss characteristic in the dif- rent diseases. These forms of generalized pigmentation are exag- rated in the areas which are normally dark, as the eyelids, the neck, e axillæ, the nipples, and the genitals. Symptomatic pigmenta- on may be very dark. In Addison's disease the patches may be- me as dark as a half-blood negro, with certain areas black, *melasma*, *melanoderma.*

Etiology and Pathology.—The causes of most of the forms of so- lled idiopathic and symptomatic chloasma have been referred to under assification. The ordinary form of chloasma is frequently unasso- ated with any demonstrable pathological condition. Where this is not e case, it is usually attributable to pregnancy or some utero-ovarian sturbance. It is a condition not uncommon in women, but one rarely en in men.

Anatomically, the lesions of chloasma do not differ from freckles cept in size.

Diagnosis.—Chloasma should not be confused with any other condi- on, although as a matter of fact it may be mistaken, on careless ex- nination, for fungus disease of the skin producing brownish discolora- ons, particularly tinea vesicolor, but of course no fungi are to be found the scrapings.

Prognosis and Treatment.—The radical treatment of chloasma is directed to the removal of the underlying conditions, and is always symptomatic. Utero-ovarian disturbances most frequently have to be treated. The patches may be treated in the same way as freckles, by use of strong bichloride lotions or by salicylic acid and resorcin pastes or plasters.

III.—TATTOO MARKS AND POWDER STAINS

Along with the discolorations of the skin due to excess of pigment may be considered the stains produced by the deposit of extraneous pigmented substance in the skin. These stains are tattoo marks, powder stains and other stains due to the accidental deposit of colored particles in the skin, and argyria.

Tattooing consists in the introduction into the skin of insoluble colored substances which become encapsulated, and thus form permanent stains.

Tattooing is one of the most primitive efforts of man at personal adornment. Like many other things that have their origin in vanity, various kinds of significance are attached to the practice, but the underlying reason for tattooing, not only among primitive races, but among the civilized, rests probably upon the inherent barbaric taste for distinctive personal decoration. Among civilized peoples and among nations in a relatively low state of civilization, like the Orientals, the practice is general, and is often carried to the most extravagant extent. Among Caucasians, aside from its general use among sailors, it is largely confined to those individuals of both the lower and higher classes who readily accept anything that is bizarre or that gives them a fancied distinction.

Brault divides tattooing among primitive peoples according to its significance, as follows: First, religious tattooing, as in the priests among the Polynesians; second, ornamental tattooing, seen in Algerians, and in the inhabitants of Oceania and Japan; third, therapeutic tattooing, practiced in Tunis, in Egypt, and in the Congo region; fourth, distinctive tattooing, practiced among the Arabs and negroes of Africa, for the purpose of defining not only different tribes, but also certain callings; fifth, obscene tattooing, which is found only rarely among savages, but which is very common among sailors and criminals.

Designs of the most elaborate character are often seen in tattoo marks, many of them showing some artistic taste and considerable technical skill. The extent to which tattooing has been carried in some individuals has been limited only by the cutaneous surface. In the well-known case of the tattooed man from Burmah, illustrated in Hebra's " Atlas," the entire surface was covered by tattoo marks. Numerous other cases of almost as great extent have been reported.

The usual method of tattooing is first to outline upon the surface the design, and then to prick out this design with a needle or a bundle of needles, and after that to rub in the pigments. For dark blues and blacks carbon in some form is used, charcoal, lampblack, India ink; other pigments used for various colors are cinnabar, carmine, indigo, and Prussian blue. The dangers of tattooing in the hands of unskilled persons by whom it is usually practiced are by no means small. All sorts of infections are possible: lymphangitis, erysipelas, chancroid, tetanus, tuberculosis, leprosy, and syphilis. Many cases of syphilis transmitted by this method are recorded in literature. The means of transmission—by the saliva of the operator, the use of an infected needle, subsequent infection of the unhealed wound—are manifest.

Practical uses of tattooing are very limited. As a means of identification tattoo marks are of course valuable, and the tattooing of habitual criminals has been suggested as a means of their ready identification. Several years ago De Wecker suggested the tattooing in black of leukomatous areas on the cornea. Very recently the highly artificial suggestion has been made of tattooing the flush area of the cheeks to represent a healthy blush. It is interesting to try to imagine how this "healthy blush" would appear on the faded skin of later life.

Powder stains, coal-dust stains, and similar stains produced by the accidental embedding in the skin of particles of colored substances, usually carbon, are in all essential characteristics identical with tattoo marks. One form of accidental marking of the skin to which attention should be called is the whitish marks which occasionally result from the precipitation of lead in the tissues during the use of subacetate of lead solution or lead-and-opium wash upon superficial wounds involving the connective tissue. The danger of these stains on the cornea from the use of subacetate of lead in the eye is well known.

Histology.—Microscopal examination of sections from tattoo marks shows that they consist of relatively large particles of pigment, situated part of them in the corium, but the larger part in the subcutaneous connective tissue. Particles of pigment are found also in the contiguous lymphatic ganglia.

Treatment.—Much ingenuity has been exercised in attempts to remove these marks. The treatment of powder stains and similar stains is largely a matter of mechanical removal, and to be successful this must be done immediately after the production of the marks, before the particles of pigment have become so disintegrated that their mechanical removal is impossible. The individual particles of pigment have to be patiently picked out, for which purpose an iris needle or a small sharp-pointed knife is most convenient. The method requires great patience both on the part of the operator and on that of the patient, but if thoroughly done immediately after the injury it gives satisfactory results. A certain amount of anæsthesia may be obtained by the application of small quantities of weak cocaine solution or by the use of an ointment of ten- to twenty-per-cent orthoform in lanolin. In connection with the mechanical removal of the particles of pigment, the use of a strong solution of H_2O_2 as a bleaching agent has been suggested, and it is perhaps the best antiseptic for use in these cases; but it is hardly possible that powder stains could be bleached by this means, since at the body temperature carbon (which causes most of the discoloration) cannot be bleached with oxygen.

The principle of almost all of the methods for the removal of tattoo marks is their destruction by mechanical means or by the production of a destructive inflammatory process which causes a superficial eschar. Very small stains can be destroyed by the use of the cutaneous punch or by electrolysis or by excision. In using electrolysis the needle attached to the negative pole of a battery with a current of from two to ten milliampères is inserted at various points around the periphery of the marks, and a sufficient reaction is produced to cause the destruction of the involved tissue. In a few days after the application a dry superficial eschar forms, which is thrown off, leaving a white scar. Of course these mechanical methods can only be applied to very small lesions on account of the scars which they produce. The various methods for the treatment of larger lesions depend upon the application of some chemical irritant which sets up an acute inflammatory process sufficiently intense to cause destruction of the superficial layers of the skin. Many irritants have been suggested for this purpose: chromic acid, carbolic acid, acetic acid, tincture of cantharides, potassium hydrate, etc. The two methods of treatment after this principle which have been most definitely worked out are those of Variot and Brault.

Variot's plan of treatment, according to Brocq, is as follows: First, he places on the tattoo marks a concentrated solution of tannin, and tattoos this in. Then a silver-nitrate pencil is rubbed vigorously over the surface. The action of the silver nitrate is allowed to go on for some moments until the surface becomes black from the formation of silver tannate in the superficial layers of the skin. In the next few days a slight inflammatory reaction occurs, and over the surface treated a closely adherent dark crust forms. After the third or fourth day there is no pain except when there is movement of the muscles under a large crust. Occasionally there is a little suppuration under the crusts, but if secondary infection is avoided no severe inflammation occurs. After fourteen or sixteen days the crust comes off spontaneously, the corium and the epidermis underneath have been repaired, and the locality of the mark is recognizable only by a superficial pink cicatrix which gradually becomes of normal color. A couple of months after the operation the scar is hardly noticeable.

In Brault's plan the irritant used is a solution of chloride of zinc, 30 grams, to 40 grams of water. The mark is tattooed with needles dipped in this solution, and in addition the surface is lightly moistened with the same solution after the tattooing. A mild inflammatory reaction is produced, followed by the formation of a crust which subsequently exfoliates, leaving a pinkish, slightly scarred surface similar to that after Variot's operation.

In the use of either of these methods several attempts may be necesary. The surface treated at one time should not exceed one or two quare inches, and of course ordinary surgical precautions as regards the leanliness of the surface, both before the operation and during the subidence of the inflammatory process, should be observed. Both of these methods are founded upon correct pathology and are worthy of trial. Variot's method would seem the one of preference, as the action of silver nitrate can be more accurately controlled than that of zinc chloride.

Ohmann-Dumesnil has proposed a method of removing these marks by digesting with digestive ferment the connective tissue which encapsuates the pigment particles and thus liberating them, so that they can be carried away by the lymphatics. For this purpose he uses glycerole of papoid or of caroid. His method is as follows: The skin is made surgically clean and then anæsthetized with a spray of ethyl chloride. The surface is then covered with glycerole of papoid or of caroid and tattooed with a bunch of six to ten very fine needles which have been dipped in the solution. The tattooing should be firmly done, but the needles should be driven just far enough to draw the least possible blood. Glycerole of papoid or of caroid is then poured over the area and it is covered with gauze. On the removal of this after two or three days the tattoo marks present a hazy appearance. Shortly after, a crust forms which later falls off, and with it the marks disappear. If any trace remains the process is repeated. This idea is ingenious, but the results have not been satisfactory in the hands of some workers. It is questionable whether the results obtained are not those due simply to a destructive inflammatory process, as in the other methods.

IV.—ARGYRIA

A staining of the skin which is essentially of the same character as tattoo marks, but which is produced from within, is argyria. In this there is, from the long-continued presence of silver in the body, a precipitation of silver in the corium and subcutaneous tissue, and perhaps in other tissues. This results in a bluish-gray or slate discoloration of the skin which is permanent. Its first manifestation is in the form of a blue line at the edge of the gums, and its development is very gradual. In well-developed cases the skin becomes of deep bluish slate color. The discoloration is universal, but is most marked in the parts exposed to light.

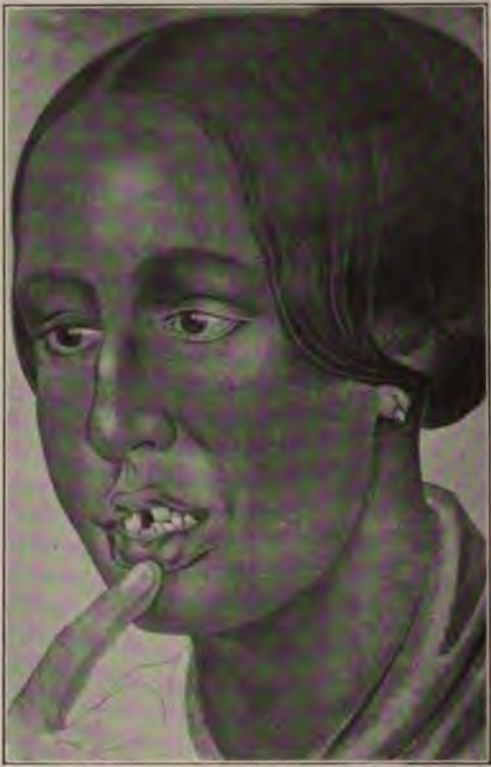

FIG. 257.—ARGYRIA.
(Photograph of plate in Kaposi's Handatlas.)

Etiology and Pathology.—The condition was much more common when nitrate of silver was commonly used in the treatment of epilepsy, but is occasionally seen now. According to Krahmer, the minimum amount which has produced argyria is 450 grains, but apparently in some cases a much smaller amount has produced it. I have had a very marked case of argyria which was produced by the daily application for a little more than a year of a saturated solution of nitrate of silver to a few patches, about a square inch in total area, of leukoplakia buccalis. The patient had never, so far as he knew, taken silver internally.

The condition is produced by the precipitation of metallic silver in the tissues. The deposit of silver particles is abundant in the subcutaneous tissue and corium, especially in the upper part of the papillary layer. It is also found in the mucous membranes, and even in the internal organs.

Diagnosis.—The slaty blue, diffuse discoloration of the skin and mucous membranes which is not affected by pressure is practically pathognomonic, although, according to Crocker, the bronzing of the skin seen in diabetes mellitus and hypertrophic cirrhosis of the liver may assume a slaty color which is indistinguishable from argyria.

Prognosis and Treatment.—When once established, the condition is permanent and cannot be removed. Yandell reported two cases in which it practically disappeared when the patients were treated for syphilis with mercurial vapor baths and potassium iodide, but the treatment has subsequently proved ineffective.

B. DIMINISHED PIGMENTATION

Lack of pigment in the skin may be either confined to limited areas or universal, and it may be either acquired or congenital.

The acquired condition is called leukoderma. This is usually confined to limited areas, and rarely becomes universal.

Congenital lack of pigment is called albinism. This may be either confined to limited areas, partial albinism, or universal, complete albinism.

I.—LEUKODERMA

SYNONYMS: *Vitiligo, Leukopathia, Acquired Leukasmus, Acquired Leukopathia, Acquired Achroma, Acquired Piebald Skin.*

Leukoderma is an affection characterized by the appearance of nonpigmented areas in the skin.

Symptomatology.—The only changes in the skin are those produced by the disturbance in the formation of pigment. There are no textural changes, and no changes in sensation. The affected areas are milky white in color, of irregular or roundish shape, and are surrounded by a border in which there is increased pigmentation, so that the white patches stand out in greater contrast. The hairs in the affected areas are usually white, although they may retain their normal color. The patches may be of any size from minute white spots up to large, irregular areas. There may be only one patch, but usually there are several, and occasionally they are so numerous as to involve the larger part of the surface of the body. The areas of predilection of the patches are the backs of the hands, the face, the neck, and the arms. The distribution of the lesions usually shows a rough symmetry, but this may be lacking.

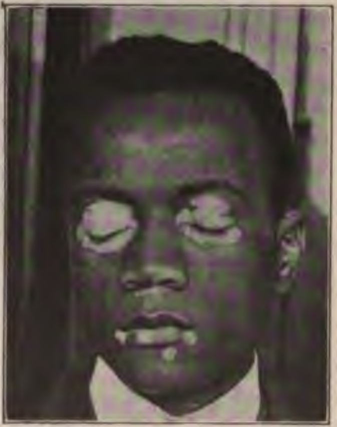

FIG. 258.—LEUKODERMA. (Heidingsfeld's collection.)

According to many authorities, the first change in the development of a patch of leukoderma is an increased formation of pigment producing a brownish patch in which a roundish white spot or spots appear later and enlarge slowly, pushing the pigment as it were before them. When the condition first comes under observation, however, it usually consists of well-defined white spots or patches which tend to enlarge gradually and afterwards become stationary. After the development of a very few patches no further evidence of the affection may become manifest. In other cases new patches appear from time to time, and as they enlarge and coalesce with other patches the disturbance becomes generalized. In very rare cases it becomes universal. The course of the affection is extremely slow, patches perhaps appearing for months or years. There may be at times apparent improvement from the general fading out of the excessive amount of pigment around the borders.

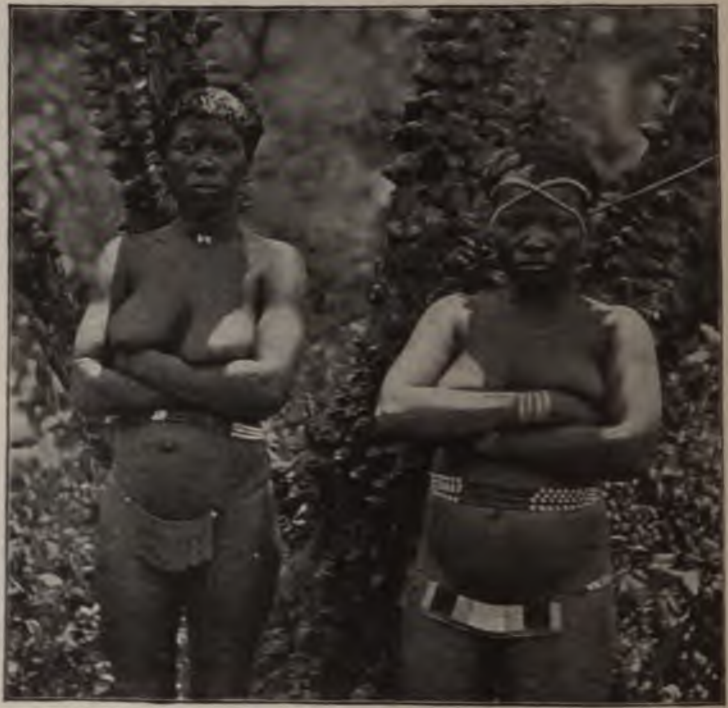

FIG. 259.—LEUKODERMA. (Shepherd, Morrow's System.)

The depigmented areas do not change. The pigment-forming tissues have apparently lost their function of making pigment, and the roduction of pigment cannot be stimulated in any way. The patches, herefore, do not tan on exposure to sunlight.

Etiology and Pathology.—Small patches of leukoderma are not ncommon. Extensive cases are very rare. The affection is commoner the dark races.

The cause of the affection is entirely unknown. It is presumably a ophoneurosis, and is frequently associated with neurotic disturbances. rarely appears before ten or after forty years of age. In some cases ere is evidence of hereditary influences.

Leukoderma is not infrequently associated with alopecia areata and casionally with morphea, both of which are probably trophic disturb-ices, and at times it is seen with other skin diseases, like psoriasis rocker). It is at times associated with systemic disturbances; mi-aine and retinitis pigmentosa (Crocker), toxic neuritis (Emery), alaria, scarlet fever, typhoid, Addison's disease, and Graves's disease. has developed after hysterectomy, after sunstroke, and after exposure extreme cold.

Anatomically the lesions show absence of pigment in the patches and crease of pigment around them. According to Marc there is thinning the rete and entire absence of chromatophores.

Diagnosis.—The simple absence of pigment from areas, with in-ease of pigment at the borders, but without other changes, is character-tic of leukoderma and does not occur in any other affection. In partial binism the fact that the patches are congenital distinguishes them from koderma.

Leukoderma in times past has been confused with anæsthetic lep-sy, and the mistake is still made. In leukoderma there are none the disturbances of sensation and none of the other well-marked anges which are characteristic of leprosy. Morphea may resemble koderma, but there is change in the texture of the skin in mor-ea; it is either dense or ivorylike, or it is parchmentlike. In mor-ea there are also dilated blood vessels to be found in the areas, and ound the borders there is a lilac instead of a brown zone. The tches of leukoderma are permanent, and little can be done to improve em.

Prognosis and Treatment.—Internal treatment is ineffective, al-ough arsenic, pilocarpine, thyroid extract, and suprarenal extract have been recommended. Locally, galvanism, with the negative pole over e patch, is recommended, but is not likely to prove of any value. The tches may be rendered less noticeable by treating the borders for the moval of pigment in the same way that patches of chloasma are treated. he white areas may be disguised temporarily by staining faintly with lute solution of walnut juice or chrysarobin.

II.—ALBINISM

SYNONYMS: *Congenital Leukoderma, Congenital Leukasmus, Congenital Leukopathia, Congenital Achroma.*

Characteristics.—Albinism is the congenital absence of pigment in the skin. It may be confined to limited areas, or it may be complete, involving the skin in its entirety, the eyes, and the hair. In partial albinism, patches of white or pinkish skin are present at birth. They are well defined, of circular or irregular shape, and may occur on any part of the body. They are most frequently found on the scalp, face, breasts, and genitals, and are often symmetrical. Where the patches occur upon hairy surfaces, the hairs are white. In albinos—i. e., those who have complete albinism—the skin is abnormally white, pinkish where vascularity is greatest, and there is no tanning on exposure to sunlight. The hair is silky and white, occasionally with a yellowish tinge. Folker describes a case of an albino with red hair. From absence of pigment in the choroids and irides, the pupils are red and the irides pink, and there is always, from excess of light reaching the retina, photophobia. Squire has reported a case in which the eyes were dark blue and there was no photophobia. According to Boyle's observations among the albinos of Borneo, universal albinism may occur in which there is not entire lack of pigment. He has recorded the case of an albino whose skin was of dirty white color and whose eyes were blue. The patient had brothers and sisters who were perfect albinos.

Etiology and Pathology.—Albinism occurs more frequently in the dark races than in whites. It is not uncommon in animals. Complete albinism usually shows a marked family tendency. It rarely occurs in all the members of a family, but may occur in several. Aubé has called attention to the influence of consanguineous marriages in the causation of albinism. In a few cases which I have been able to investigate there has been a history of marriage among blood relations, and in one group of cases the influence of this factor seemed to be beyond doubt. This factor probably explains the endemic occurrence of albinism which has been sometimes observed.

The only anatomical change in the skin is the absence of pigment.

Treatment.—No treatment is of any avail in either partial or complete albinism.

SECTION XII

NEUROSES[1]

In the group of neuroses of the skin are included the conditions rhich are characterized by sensory disturbances alone. Objective sympoms are lacking, unless they are secondary, as from scratching. As a iatter of fact the neuroses of the skin are not clinical entities, but are ierely symptoms. They have already been in part considered under ubjective symptoms in General Symptomatology.

Cutaneous neuroses may consist of increased or decreased sensibility r perversions of sensibility. The sense of touch is complex, including ensibility to pressure, which may be regarded as the tactile sense proper; ensibility to temperature, which is further separated into sensibility to eat and sensibility to cold; and sensibility to pain. These three factors n the sense of touch combined form what is called the common sense of ouch. All three of them may be disturbed independently of each other, lthough as a rule the disturbance of one is usually associated with a isturbance of the other to a greater or less degree. The cutaneous euroses may be grouped as follows:

1) Hyperæsthesia, increased sensibility of the skin, including:
   - (*a*) Hyperalgesia, increased sensibility to pain.
   - (*b*) Thermalgesia, increased sensibility to heat.

2) Anæsthesia, loss of sensibility of the skin, including:
   - (*a*) Analgesia, loss of sensibility to pain.
   - (*b*) Thermanæsthesia, loss of temperature sense.

3) Paræsthesia, perversion of sensibility of the skin, including:
   - (*a*) Dermatalgia, painful sensation of the skin, painful paræsthesia.
   - (*b*) Pruritus essentialis, itching without objective lesions.
   - (*c*) Meralgia paræsthetica.

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[1] Bronson, Morrow's "System," p. 735.—Jacquet, "La Pratique."—Von Frey, *our. A. M. A.*, September 1, 1906.

A. HYPERÆSTHESIA

Hyperæsthesia covers all degrees of increased sensitiveness of the skin. It may manifest itself only in an unpleasant sensation upon contact or upon exposure to variations in temperature, or it may be so acute as to cause extreme pain from the slightest contact (hyperalgesia) or exposure to heat or cold (thermalgesia). Hyperæsthesia of the temperature sense alone is shown especially in excessive sensitiveness to cold. Excessive sensitiveness to heat is less common. Hyperæsthesia may be local or generalized. Its location may vary in functional neuroses, like hysteria, or be confined to a fixed area, as when resulting from neuritis.

It may be due to disturbances anywhere in the nervous system from the nerve centers to the terminal nerve filaments, and is a symptom of various nervous diseases. It is perhaps most frequently associated with hysteria. It is sometimes seen before the appearance of cutaneous eruptions, usually of vasomotor origin, like urticaria, herpes zoster, herpes simplex. Hyperæsthesia is nearly always a symptomatic disturbance. In rare cases its cause is unascertainable, and it is then called idiopathic.

B. ANÆSTHESIA

Anæsthesia may be complete or partial (hypæsthesia). It usually involves temperature, touch, and pain sensibility, but these may be affected to different degrees. Analgesia may occur without loss of tactile sensibility, or both loss of sensibility to pain and temperature may occur without loss of tactile sensibility, as in syringomyelia and anæsthetic leprosy. On the other hand, there may be anæsthesia with pain, as in the anæsthesia dolorosa of Romberg. Most frequently in anæsthesia there is loss or dulling of all forms of sensibility. Anæsthesia, like hyperæsthesia, is usually limited, but may be general. It may be symmetrical or unsymmetrical, be confined to a fixed area or vary in its location, all of these features depending upon the underlying conditions producing them.

Anæsthesia may be due to causes affecting either the nerve centers, the peripheral nerves, or the intermediate nerve trunks. It occurs in the course of various nervous diseases accompanied by destructive nerve lesions either in the nerve centers or in the nerve trunks. It is seen most frequently in hysteria, in which it is usually unilateral and frequently changeable in location. It may be produced by various drugs given internally, as cannabis indica, opium, and by general anæsthetics. It may be produced by causes acting on the terminal filaments of the nerves, like old or mechanical pressure, as from the injection of water or other nert liquids into the substance of the corium, or by the action of chemical anæsthetics such as carbolic acid, cocaine, orthoform, and aconite.

C. PARÆSTHESIA

The paræsthesiæ are the perversions of the sense of touch, which may involve any or all of its factors. There may be a disturbance of the touch sense proper, so that a false impression is received of objects touched. There may be perversion of the sense of localization, so that a touch sensation at one point is incorrectly referred to the opposite side. There may be perversions of the thermal sense, so that a heated object gives the impression of cold, and *vice versa.* All of these forms of paræsthesia are symptomatic of nervous diseases.

The commoner paræsthesiæ consist in perversions of the normal sensations of the skin, showing as sensations of titillation, formication, dermatalgia, or pruritus. These are for the most part true cutaneous neuroses, produced by disturbances primarily involving the terminal filaments of the nerves of the skin.

PRURITUS[1]

Pruritus is a form of paræsthesia which is symptomatic of most inflammatory dermatoses. There are also certain affections of the skin in which itching is the essential symptom and the skin is otherwise apparently normal, except where it shows secondary symptoms as the result of scratching; these affections constitute the group which are included under pruritus as a neurosis, or

I.—ESSENTIAL PRURITUS

In association with essential pruritus there may be other forms of paræsthesia, like tingling, creeping, and burning sensations, but itching is the dominant sensation. The intensity of the itching varies greatly. It may simply cause slight annoyance at bedtime, or it may amount to almost constant torment. Like other forms of pruritus, essential pruritus is paroxysmal. It often seems to come on in waves, and is usually worse at night. In rare cases the itching may be practically constant. Frequently the waves of itching develop without apparent cause, but in all forms of essential pruritus there is hyperæsthesia of the skin and attacks of itching are excited by external impressions, like variations in temperature, friction, pressure, and other forms of external irritants. In many cases the hyperæsthesia is so acute that external impressions which in normal individuals would be hardly apparent excite severe attacks of itching. Mental impressions may also produce the attacks. It is very common to see them brought on by temporary emotional disturbances, like excitement, anxiety, or fear.

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[1] Bronson, "Etiology of Itching," *Med. Record,* October 24, 1891.—"Itching; Its Occurrence Both as a Concomitant and Cause of Disease, and Treatment," *Med. News,* April 18, 1903.—McCall Anderson and Brooke, "The Pathology and Treatment of Pruritus," *Brit. Jour. Derm.,* 1895, pp. 292 and 294.

Essential pruritus may be either universal (generalized) or localized. The term universal pruritus is applied to those cases in which the pruritus is generalized rather than where it is universal. Localized pruritus refers to those forms in which the pruritus is confined constantly to definite areas, like the genitals or anus.

UNIVERSAL PRURITUS

Universal pruritus is the form in which the itching occurs over widely distributed areas, but it is rarely if ever equal and universal over the entire body. Any part of the surface may be affected. As a rule it is worse on the legs and hands and feet. The face and scalp usually escape, although occasionally itching of the nose and ears is pronounced. Universal pruritus is not, as a rule, intense enough to cause excoriations from the scratching, but it frequently interferes with sleep, and in some cases is so persistent that the patient keeps up constantly a light rubbing or scratching of the skin. I have seen this continued so long that the patient's finger nails were worn to the thinness of paper, and the free borders were entirely worn away, although the skin itself showed no evidence whatever of scratching. In nearly all cases of universal pruritus the skin is usually dry and thin and irritable, and may show slight branny desquamation on the legs.

According to its etiology, universal pruritus may be divided into the following forms:

(1) Symptomatic universal pruritus.
(2) Pruritus from congenital cutaneous hyperæsthesia.
(3) Winter pruritus.
(4) Bath pruritus.
(5) Senile pruritus.

The first of these, symptomatic pruritus, is produced by various constitutional disturbances which reflect themselves in the skin, and is more or less temporary. The others are essentially due to a permanent condition of hyperæsthesia of the skin, which makes it more than ordiirily irritable to all forms or to some peculiar form of external irrita- n. Winter pruritus and bath pruritus, I am inclined to believe, are many cases due to congenital hyperæsthesia. Senile pruritus is a con- tion of hyperæsthesia acquired in old age.

Symptomatic universal pruritus is produced by practically the me causes which produce urticaria. Indeed, attacks of general pruritus t infrequently precede attacks of urticaria, and in some cases alternate th them. Like urticaria, universal pruritus may be produced by emo- nal disturbances, by functional and organic nervous diseases, but its st frequent cause is toxic disturbances. It may be produced by inter- l administration of various drugs, notably opium; or by the use of same sort of foods that produce urticaria. A very severe and per- tent form of it is produced at times by jaundice, and other toxic forms ur as result of various gastrointestinal disturbances, especially those ociated with constipation. The gouty or rheumatic diathesis, nephritis, d diabetes may also produce it. The duration of all these forms of nptomatic pruritus depends upon the duration of the exciting cause. rely are they permanent.

Pruritus from Congenital Hyperæsthesia.—So far as I know, this rm of pruritus is not described, but I have studied carefully two cases ich I believe are to be attributed to this cause alone. The patients d been from earliest childhood subject to severe itching on exposure the body to air, from the irritation of bathing and of clothing, and pecially from that produced by getting warm in bed. The patients so showed dermographism, but the itching occurred entirely independ- t of any appearance of wheals. These cases are to be regarded only extreme examples of the irritability which exists in varying intensity all individuals.

Winter Pruritus (*Pruritus hiemalis*).—Somewhat analogous to the ses of congenital hyperæsthesia of the skin are the occasional cases winter pruritus. This consists simply in the itching of the skin which produced by cold weather. It comes on with each autumn and con- nues to cause annoyance until late spring. It is most severe upon un- essing at night. Upon exposure of the surface to the air there is an aggerated appearance of goose flesh, and the itching continues for an ur or two after retiring. It usually disappears during the day. In e severer cases there may be evidences of scratching. In its slighter rms winter pruritus is exceedingly common. The severer cases, in hich the condition amounts to an affliction, are uncommon.

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[1] Duhring, *Phila. Med. Times*, January 10, 1874.—Hutchinson, *Brit. Med. Jour.*, 75, vol. ii, p. 773.—Morago Porras, *Trans. Internat. Cong. Derm. and Syph.*—Cor- t, *Jour. Cutan. Dis.*, 1891, p. 41.

Summer Pruritus (*Pruritus æstivalis*).—Hutchinson has described a summer pruritus developed by the heat of summer, which is apparently analogous to winter pruritus, but, as Bronson says, "this is not generally recognized in this country." The itching common in summer is nearly always the result of dermatitis produced by sweating or of analogous conditions.

Bath Pruritus[1] is a form analogous to winter pruritus, with the difference that it is excited by bathing while winter pruritus is excited by exposure to cold air. It is, like winter pruritus, a manifestation of the hyperæsthetic skin. The essential feature of it is the occurrence of itching after bathing. This is frequently so severe as to cause the patient to dread a bath. It may continue only a few minutes after emerging from the bath, or may be annoying for an hour or more. According to Stelwagon, it is usually more persistent and severe when the patient goes to bed after the bath, and disappears more quickly if he immediately dresses.

Senile Pruritus is a not uncommon form of essential pruritus which occurs as an accompaniment of the senile changes of the skin. The itching may cause the patient extreme annoyance, but it is rarely severe enough to cause the production of excoriations. It is usually most pronounced on the legs, is worse at night, and is excited by rough clothing. The condition is worse in winter, although attacks of itching are excited in winter by exposure to heat, so that the victims prefer to be in a cool atmosphere and sit as far as possible from any source of heat. Senile pruritus is usually seen in thin, delicate skins, and frequently appears in persons who show little or no traces of senile keratosis or other degenerative lesions in the skin.

Acarophobia.—Occasionally there are seen cases of generalized pruritus in which the patients have the delusion that they are affected by microörganisms in the skin which produce their itching. This rare condition, called acarophobia, is usually seen associated with senile pruritus. The patients will show rolled-up masses of epidermis or of dirt, or other small objects which they find upon the skin, and demonstrate them as the parasites. No amount of reasoning or of assurance will disabuse them of their delusion; they are insane upon the subject.

LOCAL FORMS OF ESSENTIAL PRURITUS

Rare cases of persistent essential pruritus of the palms and soles and of the tongue are described, but the only common forms of localized essential pruritus occur about the genitals and anus.

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[1] Stelwagon, *Phila. Med. Jour.*, October 22, 1898.

Pruritus ani et vulvæ.—Pruritus of the genitals, perineum, and anus frequently occur together, and have many features in common. Vulvar pruritus is usually associated with more or less anal pruritus; anal pruritus in males, however, is not associated as a rule with genital pruritus. Pruritus ani and pruritus vulvæ may be trivial affections consisting in occasional attacks of itching, usually most marked at night. In their severer forms they are most distressing affections, interfering with comfort by day and often causing almost intolerable itching at night, which reatly interferes with sleep. The itching is nearly always paroxysmal. There may be hours of entire freedom, interspersed with attacks of ching; these are especially severe on going to bed and during the ight. Pruritus vulvæ usually affects all of the external genitals and he introitus vaginæ. It may be confined to the labia, the clitoris, r other parts of the external genitals. In the severer forms of essenal pruritus about the anus and genitals there is usually secondary czema produced by scratching. The parts are excoriated, the skin macerted and whitish, and often there is considerable induration as a result f a chronic dermatitis. In such cases there is often profound impreson on the patients' nervous systems. They are oppressed to a point where life is almost unbearable, and would accept relief at almost any ost.

Both pruritus vulvæ and pruritus ani most frequently occur in middle r old age.

According to Crocker, pruritus ani in adults is nearly always due to epatic derangement, and it is frequently associated with hæmorrhoids nd other evidences of hepatic disturbance, but disturbances of the liver are only a part of the causes of the affection. It may be due to any condition, like tumors, causing congestion of the pelvic viscera. In many cases it is produced by intestinal catarrh, and by the toxic disturbances associated with intestinal fermentation. It may be due to the gouty diathesis, and, if not produced, it is undoubtedly exaggerated by he habitual use of narcotics and stimulants, especially tobacco, alcohol, coffee, and tea. The presence of fissures, fistulæ, and hæmorrhoids s in some cases the exciting cause of the affection, and cure of these esions is followed by relief. In a good many cases no definite cause can be assigned, and we have to fall back on the assumption of a pure neurosis, an assumption which is usually fortified by the neurotic character of the patients.

Pruritus vulvæ may be a sequence of pruritus ani, and may be produced by the same causes. It may also be due to uterine or ovarian disturbances, to acute or, more frequently, chronic vaginitis or urethritis. It frequently occurs with diabetes, but then is associated with eczema. It is usually exaggerated during the menses. It is apparently in some cases a pure neurosis, especially when developing, as it frequently does, during the climacteric.

Pruritus scroti.—Occasionally essential pruritus of the male genitals occurs of similar character to pruritus vulvæ. This is usually confined to the scrotum, but it is uncommon.

Rarely in both males and females there is pruritus of the urinary meatus. In most cases this is due to local irritation, but in some cases it is an essential pruritus, usually the result of some irritation in the urethra or at the neck of the bladder, like urethritis, stricture, stone, or cystitis.

Pruritus ani and pruritus vulvæ are not infrequent in children, as a result of intestinal worms, or occasionally as a result of intestinal catarrh. In infants the affection is usually trivial, not intense enough to cause excoriations from scratching. There is a popular impression that itching of the nose in children is also caused by intestinal worms. Personally I believe that the tendency to pick and scratch the nose in infancy is altogether independent of association with intestinal worms.

DIAGNOSIS OF ESSENTIAL PRURITUS

The diagnostic feature in all forms of essential pruritus is the existence of itching without other apparent changes in the skin. After this is established, the much more difficult question to determine is the cause of the pruritus. In determining this, the factors already referred to under the etiology of the various forms of pruritus have to be carefully considered.

The establishment of the diagnosis of essential pruritus is one which does not often have to be made except in pruritus of the genitals and anus, but it is one in which there is little reason for mistakes unless the observer is careless in overlooking parasites or easily recognizing objective symptoms. The condition which is most likely to be confused with essential pruritus is urticaria. Next after this comes pediculosis and scabies, and then various forms of dermatitis. None of these should cause confusion if the observer takes the pains to look for common objective symptoms.

Pruritus of the vulvæ or anus has to be diagnosticated from pruritus due to eczema or other forms of inflammatory dermatoses of these parts. Essential pruritus is usually more severe, and has a history of longer duration, and is more intractable to local treatment. The relief of the secondary eczema is not followed by the relief of itching which takes place when the itching is the result, and not the cause, of the eczema. Itching of the anus and vulvæ in infants is much more frequently a result of dermatitis from irritating secretions than it is a form of an essential pruritus.

PROGNOSIS AND TREATMENT OF ESSENTIAL PRURITUS

Generalized pruritus, which is symptomatic of toxic disturbances or of other systemic conditions, is benefited by treatment directed to the underlying condition. In general, the internal treatment of such cases is along the same lines as that of urticaria. In cases of symptomatic essential pruritus, it is desirable to stop as far as is practicable the use of alcohol, tobacco, coffee, and tea, and to interdict the use of hot liquids and seasoned foods, and to have the diet simple. Free elimination should be provided. Water should be drunk abundantly, and in many cases saline aperients are indicated. Sometimes these cases are benefited by internal use of remedies directed solely to the relief of itching. Of these, pilocarpine in my experience is most efficient, given hypodermically in doses just sufficient to produce slight perspiration or salivation—usually $\frac{1}{15}$ to $\frac{1}{10}$ grain of pilocarpine hydrochlorate or nitrate; to be repeated after the effects wear off. Bulkley highly recommends cannabis indica as a sedative, in doses of 30 minims t.i.d., after meals, well diluted with water. Schamberg recommends carbolic acid internally in liberal doses. Wine of antimony, 5 to 10 drops t.i.d., is recommended by Hutchinson in senile pruritus. In extreme cases temporary benefit may be obtained by the use of such sedatives as the bromides, chloral, sulphonal, or phenacetin. Morphine, unless given to the point of producing narcosis, is not likely to stop the itching and may increase it, so that it is not ordinarily available for the relief of pruritus.

Winter pruritus, bath pruritus, and senile pruritus are all essentially forms of hyperæsthesia of the skin, either congenital or acquired, which are little or not at all influenced by internal measures. Any rational indications which may possibly exist should be treated symptomatically, but the treatment of these forms of pruritus is practically altogether by local measures. By local treatment the itching can be temporarily relieved.

The local treatment of the various forms of general pruritus consists in the first place in avoiding as far as possible all forms of local irritation. Among these are rough underclothing, soap and water, and exposure of the body to cool air. The underclothing should be as smooth and unirritating as possible; light cotton underwear, or better, light linen or silk next to the skin. In all cases frequent bathing with soap and water is irritating, and should not be indulged in beyond the point necessary for cleanliness. The bath may be made more bland by adding bicarbonate of soda or bran or starch. After bathing, the body should be powdered either with talcum or starch or some other inert powder. In many cases the greatest comfort is gotten from anointing the body after the bath with a bland oil, such as olive oil or liquid vaseline.

In bath pruritus the necessary bathing should always be done in tepid water, neither hot nor cool, and the bath is less irritating if it is supplemented with an alkali or a demulcent like bran or marshmallow. The bath should be taken in as short a time as possible, the body dried by tapping the surface—not by rubbing—and this should be followed by the use of a dusting powder or a bland oil. If the patient immediately dresses the itching is less than if he gets into bed after bathing.

Positive applications for the relief of the itching of essential pruritus are found in antipruritic drugs. Of these carbolic acid and menthol are most useful. A good antipruritic oil is made from olive oil or liquid vaseline containing either one-half- to one-per-cent carbolic acid, one- to five-per-cent menthol or thymol, or one- to two-per-cent salicylic acid or resorcin. These may also be used in the same strengths in the form of ointments with vaseline, or rose ointment, or ointment of zinc oxide as the base. An excellent application is a calamine liniment containing one-half- to one-per-cent carbolic acid or menthol. Another good application, which is very convenient because it is not greasy and disagreeable, is tragacanth liniment containing one-half- to one-per-cent carbolic acid. As a rule, oily or demulcent applications are better borne than drying applications, but occasionally, especially in senile pruritus, the use of lotions is most satisfactory. Of these calamine lotion containing one-half- to one-per-cent carbolic acid is a type of the most useful. Other useful lotions are distilled extract of witch-hazel or water with twenty-five-per-cent of alcohol or spirits of cologne. To these may be added carbolic acid, one half to one per cent; resorcin, one to five per cent; or compound tincture of coal tar, five to ten per cent. In some cases corrosive sublimate, 1 : 500 or 1 : 1,000, in water and alcohol is a good application.

In treatment of pruritus of the anus and genitals every possible effort should be made to get rid of underlying causes. Fissures should be treated, and gastrointestinal and metabolic disturbances corrected if possible. Pathological conditions in the genito-urinary tract should be looked for, and if possible corrected. But after all of these general measures have been attended to, it must be confessed that in many cases the affection still persists, and we are reduced to symptomatic treatment.

The local treatment of these cases is directed primarily to the relief of itching. In the start it may be necessary to treat secondary eczemas by soothing applications, but, as a rule, if the itching can be controlled and the scratching stopped, the eczema takes care of itself. Occasionally these patients need sedatives upon retiring to get them to sleep. For this purpose a suppository containing one half to one grain of extract of belladonna may be useful, or other sedatives may be given with caution.

In both pruritus of the anus and of the vulva scrupulous attention should be paid to cleanliness. The parts should be bathed frequently ough to keep them free from all secretions; this bathing is not, as a le, irritating. Indeed, one of the best applications to relieve the itching water, as hot as can be borne, dabbed on the parts for several minutes a time.

Among drugs for the relief of itching about the genitals and anus, bolic acid and menthol, in the strength of one to five per cent or more, most useful. They may be used in lotions or in ointments, and fre- ently they may be borne with comfort in strengths which would prove hly irritating to inflamed surfaces. In severe cases cocaine ointment the strength of one half to two per cent may give temporary relief. re prolonged relief may often be gotten from the use of strong astrin- nts or caustics. One of the best of these applications is potassium- rmanganate solution, one-half- to one-per-cent strength. Another ringent application is nitrate of silver, one to two per cent in water. ese strong applications should only be applied at intervals of several ys, and then in conjunction with ordinary antipruritic applications. measure which Crocker recommends highly is to paint the parts every ght with compound tincture of benzoin. Whatever application is ed, it is desirable to have at hand a bland ointment like oxide-of-zinc ntment, to be applied by the patient when he has excoriated the parts om scratching.

The application of static electricity or high-frequency currents some- nes gives relief, but in my experience they have been disappointing. y far the most useful agent in my experience in giving prolonged lief in both vulvar and anal pruritus is x-rays. These should not be ven to the point of producing erythema. Light exposures are given intervals of one to three days, preferably with a hard tube. Many servers testify to the prolonged relief given by the use of x-rays, and some cases cure results.

Sometimes surgical measures are tried, including multiple scari- ation with the actual cautery, and even more radical surgical meas- es, but these are not usually satisfactory.

II.—MERALGIA PARÆSTHETICA[1]

Under this term Bernhardt and Roth have described a form of ræsthesia occurring in the area of distribution of the external cuta- ous femoral nerve. About thirty-five cases only have been recorded, t the affection is not excessively rare. The disturbance is always nfined to this area, the outer side of the lower two-thirds of the igh. There is usually hypæsthesia of the affected skin, with various other disturbances of sensation, such as numbness, prickling, formication, a feeling of constriction or distention, cold or heat. Occasionally there is hyperæsthesia and pruritus. The disturbances of sensation are usually excited by walking, but may be produced by pressure and other forms of local irritation.

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[1] J. C. White, *Jour. Cutan. Dis.*, April, 1906.

Its etiology is uncertain. In most of the cases the sensations are excited by exercise, and it is probable that the affection is produced by the stretching of the nerve in the movements of the fascia lata in the area where the nerve descends through the sheath of the fascia. Most of the cases are said to occur in alcoholics. In White's case, a healthy man of fifty-five, no causative factors could be found beyond exercise.

The malady is chronic and apparently uninfluenced by treatment.

III.—DERMATALGIA

SYNONYMS: *Neuralgia of the Skin, Rheumatism of the Skin, Dermalgia.*

Dermatalgia is spontaneous pain in the skin. It differs from hyperæsthesia in that in hyperæsthesia the painful sensations are excited by external impressions, while in dermatalgia they are spontaneous, but it is nearly always associated with hyperæsthesia of the involved surface.

The pain may be burning, stinging, or shooting in character; it is usually variable, and frequently worse at night, and is likely to be excited by motion or external contact (hyperalgesia). Dermatalgia is nearly always confined to circumscribed areas. It is most common upon hairy parts. It is not usually accompanied by any change in the appearance of the skin.

Causalgia is a form of dermatalgia characterized by burning pain; it is a symptom of glossy skin, and was described by Weir Mitchell in connection with that affection.

Dermatalgia may be without ascertainable cause, but it is usually, especially in its severer grades, associated with nervous disease. Idiopathic dermatalgia frequently comes on after exposure to cold, and is often probably rheumatic. Symptomatic dermatalgia may result from hysteria, neuritis, and various diseases in which neuritis occurs, like syphilis, diabetes, and malaria. It also occurs in various diseases of the central nervous system, notably in locomotor ataxia.

The treatment of dermatalgia is symptomatic. Most frequently, when not due to neuritis or some definite nervous disease, it is a manifestation of rheumatism and is benefited by salicylates, aspirin, quinine, and the usual remedies for rheumatism. Such anodynes as aspirin, phenacetin, kryofin, often afford temporary relief. Crocker recommends counter-irritation in the form of a mustard leaf over the center from which the nerve supplying the affected part emanates.

IV.—ERYTHROMELALGIA[1]

Erythromelalgia is a form of dermatalgia accompanied by more or ss redness of the affected part. Weir Mitchell described it in 1872 d gave it the name erythromelalgia (red neuralgia).

Symptomatology.—The pain is a burning, throbbing, neuralgic in more or less paroxysmal in character. It may come on in attacks iich last from a few minutes to an hour or more, or be almost conuous. It may appear spontaneously, or be excited by external impresns. It is usually worse when the part is dependent or warm. Accomnying the dermatalgia there is a characteristic mottled redness of the ected part. This is more marked when the part is dependent, and y slowly disappear when the part is elevated. In some cases there is ssive congestion and even swelling, suggesting Raynaud's disease, and sner has reported two cases in which after long persistence of the contion gangrene occurred.

Hyperidrosis is commonly present in the affected part, and often there increased temperature.

The affection usually begins in a small area, but, as a rule, gradually tends for a while and then remains stationary. The leg and especially e foot are the parts typically affected by the condition. It may inlve one or both feet, one or both hands, or all of the extremities. In me cases it is sharply limited to a part of a hand or foot, or to one or few digits. The condition is very chronic and may exist for an infinite number of years.

The condition is in most cases symptomatic of nervous disease. It ay be due to neurasthenia, and has been known to follow severe emoonal disturbances. In a case of Mitchell and Spiller's it was associated ith peripheral neuritis, and it is probable that peripheral neuritis is ost frequently the underlying cause. It was noted in some of the verer cases of arsenical poisoning in the Manchester epidemic, in which was associated with peripheral neuritis. It is also found in conction with various brain and cord diseases, like multiple sclerosis, bes, meningitis, myelitis, and Morvan's disease. Morel-Lavallée has en it associated with a mild form of Raynaud's disease. Indeed, in me of the symmetrical cases the distinction between erythromelalgia d Raynaud's disease might be very difficult to draw.

A large majority of the cases are males, and according to Hallopeau d Laredde it is commonest in adolescence and early adult life.

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[1] Weir Mitchell, *Phila. Med. Times*, 1872, pp. 81 and 113; *Amer. Jour. Med. i.*, 1878, vol. lxxvi, p. 17.—Weir Mitchell and Spiller, *Amer. Jour. Med. Sci.*, vol. vii, 1899, p. 1 (review and bibliography).

Prognosis and Treatment.—The affection is persistent and is little influenced by treatment. General treatment is symptomatic. Phenacetin, sodium salicylate, aspirin, and the other anodynes may give some relief. Nerve-stretching in Weir Mitchell's hands produced varying results. Some benefit may result from the galvanic or Faradic current. A case of Crocker's found some relief from bathing the foot with hot water, but as a rule heat makes the pain worse, and in most cases temporary relief is obtained by use of cooling applications, such as cool bathing of the part, exposure to cold air, or the application of menthol preparations.

SECTION XIII

NEW GROWTHS—NEOPLASMATA

The group of neoplasms of the skin includes various conditions which characterized by the increase of tissue elements. It is a rather heteroeous group, and does not include all conditions which come strictly hin the term of the definition. For example, many granulomatous iors of infectious origin are grouped more logically with infectious ases, and various other tissue overgrowths with the hypertrophies.

Cutaneous neoplasmata may be divided as follows:

A. Degenerative; in which there are marked degenerative changes in tissues:

(1) Molluscum Contagiosum.
(2) Xanthoma.
(3) Xanthoma Diabeticorum.
(4) Colloid Degeneration of Skin.
(5) Colloid Degeneration of Granulation Tissue.
(6) Degeneration of Elastic Tissue.

B. Benign; tumors composed of normal tissue without a tendency to rabundant destructive proliferation. Here are included new growths ll tissues:

(1) Cicatrix.
(2) Keloid.
(3) Fibroma.
(4) Neuroma.
(5) Lipoma.
(6) Myoma.
(7) Tumors of the Blood Vessels:
    (*a*) Vascular nævus; (*b*) telangiectasis; (*c*) angioma serpiginosum.
(8) Tumors of the Lymphatics:
    (*a*) Lymphangiectasis; (*b*) lymphangioma circumscriptum; (*c*) lymphangioma tuberosum multiplex.
(9) Benign Epithelial Tumors:
    (*a*) Multiple benign cystic epithelioma; (*b*) adenoma sebaceum.

C. Malignant, tumors with a tendency to destructive proliferation:

(1) Growths of the epiblastic elements:
  (*a*) Epithelioma; (*b*) carcinoma.

(2) Growths of mesoblastic elements:
  (*a*) Sarcoma; (*b*) leukemia; (*c*) pseudoleukemia; (*d*) mycosis fungoides.

Sometimes the lines of demarcation between these subdivisions break down, so that it is difficult to class accurately a given pathological condition.

A. DEGENERATIVE NEOPLASMATA

I.—MOLLUSCUM CONTAGIOSUM [1]

SYNONYMS: *Molluscum sebaceum, Molluscum sessile, Acné varioliforme* (Bazin), *Molluscum verrucosum* (Kaposi).

Molluscum contagiosum is a disease of the skin consisting of small, waxy, rounded, epithelial tumors having a rounded opening at the apex which is filled with degenerated epithelial *débris.*

The disease was first described by Bateman at the beginning of the nineteenth century.

Symptomatology.—The lesions are pinkish, or pearly white, waxy-looking tumors from the size of a pinhead to a small pea. In very rare instances the lesions develop into tumors from one to three inches in diameter (molluscum giganteum). The smallest are usually acuminate or globular. When they become larger they are rounded and somewhat flattened. On the summit of each lesion there is an umbilicated, round opening from which a cheesy-looking substance can be expressed, but the lesion cannot be entirely emptied by moderate pressure like a dilated sebaceous gland. The number of the lesions, as a rule, varies from one or two to a dozen or more. Occasionally they may be more numerous; in rare cases they are widely distributed and amount

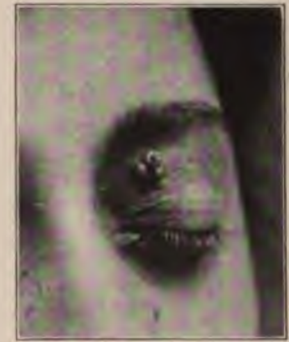

FIG. 260.—MOLLUSCUM CONTAGIOSUM. (Schamberg's collection.)

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[1] Neisser, *Archiv*, 1888, p. 553 (complete review and full bibliography).—Fordyce, *Jour. Cutan. Dis.*, 1892, pp. 367 and 372.—Stelwagon, *Jour. Cutan. Dis.*, 1895, p. 50.—Pringle, *Brit. Jour. Derm.*, 1898, p. 198.—Frick, *Jour. Amer. Med. Assoc.*, 1899, i, p. 536.—Chas. J. White and W. H. Robey, Jr., *Jour. Med. Research*, April, 1902, p. 255 (valuable review and study of histology, full bibliography).

00 or more—400 in a case of Frick's. When the lesions are multiple
may be grouped together and form semiconfluent masses, each tumor,
ever, remaining distinct. The lesions occur most frequently on the
, particularly about the eyelids and the mouth. Other common loca-
s are the breasts and the genitals. In rare cases their distribution
neral. The lesions have been observed on the mucous membranes
gue, Colcott Fox; vermilion borders of the lips, Allen). The dis-
is persistent. Lesions develop slowly, and after reaching a certain
they remain unchanged for weeks or months. New lesions occasion-
appear, so that lesions of varying size may be seen. The tumors may
me infected, suppurate, and disappear without scarring, or they may
pear spontaneously after an existence of one or two months. If
to themselves they may persist much longer—for one or two years
ore—and in some cases the lesions exist for several years. This is
icularly likely to happen in the giant lesions. In a case observed by
ter Smith a tumor three inches in diameter had persisted for thirty

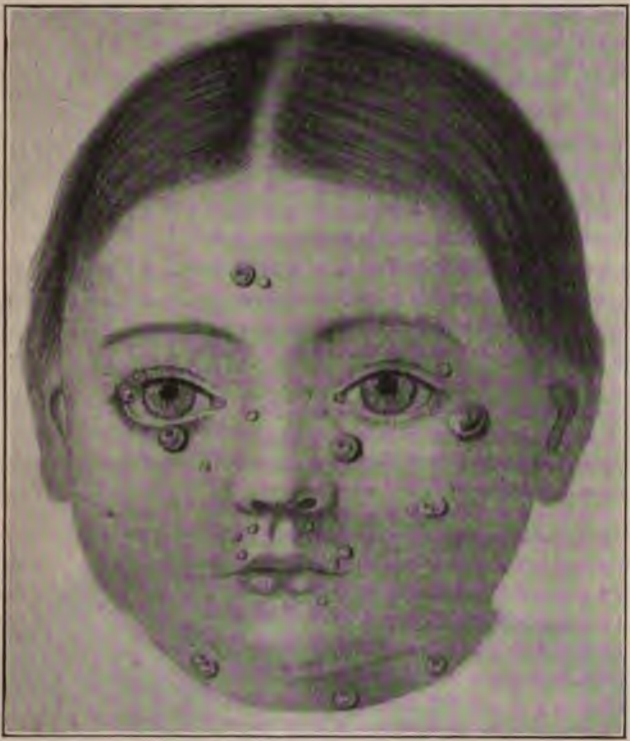

FIG. 261.—MOLLUSCUM CONTAGIOSUM.
(Photographic reproduction from Sydenham Atlas.)

years, and had not grown for fifteen. The affection is accompanied by no constitutional or subjective symptoms.

The affection may of course occur in connection with others, and as it is frequent in children in whom warts are also frequent, this association is sometimes seen. It is probably not more than an accidental association. Lieberthal[1] has noted the interesting combination of molluscum contagiosum with xanthoma.

Etiology and Pathology.—Molluscum contagiosum is rather uncommon. It is contagious, although the contagion is very weak. The disease is most frequent in children of the poorer classes; numerous groups of cases have been observed in institutions for children and in families. In England the Turkish bath seems to be a source of the affection. The disease has been observed in animals—in chickens (Colcott Fox), in mated sparrows (Shattock). Salzer has recorded a case in a woman in whom the affection was apparently contracted from pigeons, and Hutchinson a case apparently contracted from a dog. In a few cases successful inoculations have been made (Retzius, Peterson, Vidal, and others), and in a few instances inoculations have taken place in physicians from their cases (Wigglesworth, Allen, Brocq). The inoculation period is long, varying from two to nine or ten weeks or several months.

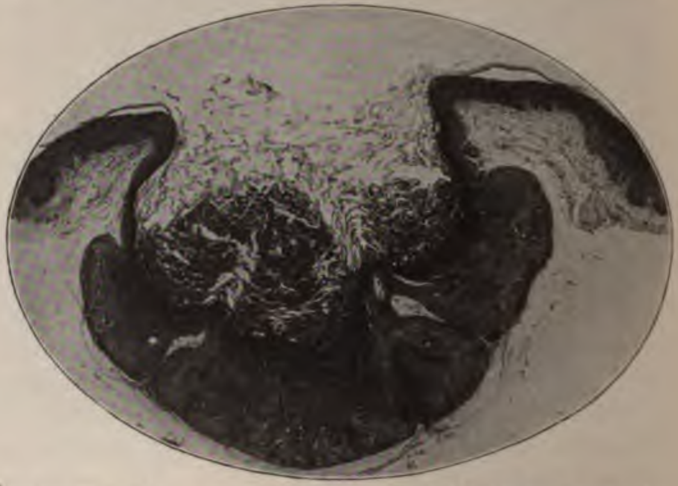

Fig. 262.—Molluscum Contagiosum. (C. J. White's collection.)

The lesions are almost surely produced by an organism, but this has not been discovered. The molluscum bodies which were regarded as psorosperms have been demonstrated by Piffard and others to be degenerating epithelial cells.

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[1] *Jour. Cutan. Dis.*, April, 1905.

The lesions strikingly suggest in their gross appearance tumors of he sebaceous glands; but it is generally accepted that they are epithelial umors independent of the sebaceous glands. They may start either n the epithelium of the hair follicles or in the rete itself. The cells f the tumors undergo a peculiar degeneration which has been regarded s a hyaline or colloid, but C. J. White and Piffard have shown hat they contain normal keratin. The growths are lobulated epihelial tumors with a constriction at the neck. They are surrounded by a fibrous capsule which sends thin, fibrous septa between the obules. Above these fibrous septa the lobules are separated by septa of keratin. The basal layer of the tumors consists of a layer of palisade epithelium, the cells over which are rounded and nodulated, but the peculiar degeneration of the cells begins very quickly, and molluscum bodies may be found very near the lowest layer. The cells rapidly become white, opaque, and homogeneous, with entire obliteration of their internal structure, increasing at the same time in size and thus forming the large, round, so-called molluscum bodies. Ultimately the cells break down and form in the neck of the tumor a yellowish, cheesy, disorganized mass.

Diagnosis.—The globular, acuminate or flattened, pinkish, paraffinlike tumors with a central opening from which a cheesy mass can be extruded without entirely emptying the tumors are characteristic. There are no other lesions like them. They slightly resemble small fibromata, milia, verrucæ, and comedones, but anything more than a superficial examination will readily detect the differences.

Prognosis and Treatment.—The affection is liable to persist indefinitely if untreated, but yields readily to treatment.

The tumors should be slit by a complete incision, which is usually painless, and the contents then pressed out between the thumb nails or with the back of a dermal curette. After this it is well to touch the cavity with tincture of iodine. Another method is to press out the soft contents and then touch the inside of the cavity with tincture of iodine on the end of a toothpick. After this the tumor gradually dries up and exfoliates.

II.—XANTHOMA[1]

SYNONYMS: *Fibroma lipomatodes* (Virchow), *Xanthelasma*, *Vitiligoidea*, *Molluscum cholesterique* (Bazin).

Xanthoma is a disease of the skin consisting of chamois-yellow plates or nodules embedded in the corium.

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[1] *Transactions London Patholog. Soc.*, 1882, p. 376.—Török, *Annales*, 1893, pp. 1109 and 1261 (review and bibliography).—Hyde, *Jour. Cutan. Dis.*, 1895, p. 183.—Pollitzer, *New York Med. Jour.*, 1899, ii, p. 73.—Whitehouse, *Jour. Cutan. Dis.*, 1904, p. 470.

Xanthoma occurs in two well-defined clinical forms: Xanthoma planum, and xanthoma tuberosum or xanthoma multiplex.

The first of these is a localized eruption around the eyelids; the second is widely distributed.

The two forms are closely alike in the histological structure of the lesions, and it is probable, on the whole, that both forms are essentially the same; clinically, however, they are sharply distinguished. There is a form of xanthoma multiplex which occurs in diabetes mellitus (xanthoma diabeticorum). This has sharp clinical differences from the ordinary cases of xanthoma multiplex, but is probably a variant of the same pathological condition.

Xanthoma planum.—Xanthoma planum occurs in the form of roughly rectangular chamois-yellow plates embedded in the corium. They are somewhat irregular in outline, from one eighth to one half of an inch in their long diameter (which is usually parallel to the margin of the lids), and are slightly or not at all elevated. The epidermis over them is normal; they are soft to the feel and without induration. Upon stretching the skin they show as distinct infiltrations which, under a hand lens, are seen to be composed of closely set aggregations of small, yellow granules, each granule having a minute, reddish central point. The usual color of the lesions is distinctly yellowish, accurately described as chamois yellow. In rare instances the lesions have a white or creamy color, and still more rarely they may be dark yellow to dark brown; in a case of G. H. Fox's, an old patch of xanthoma was so dark as to resemble an ecchymosis. Xanthoma begins as pinhead-size lesions, which gradually increase to the size of a wheat grain or larger. The lesions appear around the eyelids, and are said to begin usually on the left upper eyelid, after which they appear on the right lid. The upper lids are said to be more frequently involved than the lower. The lesions are usually multiple around the eyes, and are always roughly symmetrical. There may be only a few plates, or they may be so numerous as to encircle the lids. Adjacent plates may coalesce, to form irregular bands or patches. In the typical type of the affection the lesions are confined to the eyelids or the closely adjacent skin. Exceptionally they appear upon other parts of the face, even in the mouth. In xanthoma multiplex, plane lesions, interspersed with nodules, may occur upon other parts.

Xanthoma multiplex (*Xanthoma tuberosum*).—The lesions of xanthoma multiplex are nodular instead of flat, but otherwise their appearance is like those of xanthoma planum. The tumors are from the size of a pinhead to a small pea or hazelnut; occasionally they are as large as a walnut or a small apple. The largest lesions are usually formed from the coalescence of smaller lesions, and may be of irregular form.

They frequently show dilated blood vessels upon their surfaces. In **xanthoma** tuberosum there is a much greater stroma of connective tissue, so that the lesions are denser than the plate lesions; they may be of firm to hard consistence. In nodular xanthoma the lesions are usually numerous, and are not sharply circumscribed to any particular area. The areas of predilection are the hands and feet, including the palms, soles and the ankles, the elbows, and the buttocks. The lesions may be very abundant, and in extreme cases they are widely distributed. In xanthoma multiplex other tissues besides the skin may be involved. Lesions may occur on the lips, in the mouth and the fauces, and in the conjunctivæ and iris. In a case of mine associated with diabetes insipidus there were definite physical signs of their existence in various viscera, including the heart and abdominal organs. In a case demonstrated by Lieberthal there was a tumor the size of a small apple in the tendo-achilles, besides various other subcutaneous lesions.

Xanthoma planum is a very persistent affection. The lesions develop slowly, reaching their full size in the course of one or two years, and become numerous only after several years. They show little or no tendency to disappear spontaneously. In xanthoma multiplex the lesions develop more rapidly, and may become abundant in the course of several months. In xanthoma multiplex there is sometimes a slight tendency to involution, and in a few instances the disease has completely disappeared.

Xanthoma has no subjective symptoms, as a rule. In a few cases itching or burning has been recorded.

Xanthochromia.—Besnier and Gailleton refer to xanthoma-colored noninfiltrated patches which they have observed in cases without jaundice. The condition is most marked in the face and trunk, and is not associated with jaundice or yellowish discoloration of the conjunctivæ or mucous membranes.

Etiology.—Xanthoma planum is a rather common affection in women, but, aside from the slight disfigurement, it causes no trouble, and medical advice is not likely to be sought concerning it. It is more common in adults, and is very unusual in children. About sixty per cent of the cases are in women. It is more common in women who are spare built and not very vigorous, and in those who have pasty complexions and dark rings around the eyes. The condition is frequently associated with migraine. In Hutchinson's statistics, one half of the patients suffered from migraine and one sixth had had jaundice, but the association of xanthoma of the lids with jaundice is not so frequent as to indicate that jaundice is an important cause. Various other pathological conditions, like gout, syphilis, and uterine disturbances, are at times seen with it, but the association is probably little more than accidental. Not infrequently there is an hereditary tendency to xanthoma planum. It occurs in members of the same family and in direct descendants, sometimes skipping a generation. In two cases of xanthoma, in sisters, in my experience, it was an interesting fact that one of them suffered from migraine and the other did not.

Xanthoma multiplex is rare. It occurs in all ages and in both sexes. In adults there is usually a history of previous attacks of jaundice; this association occurred in twenty-three out of twenty-eight cases in the statistics of the Committee of the London Pathological Society. In one case in my experience, in a boy fourteen years old, it was associated with diabetes insipidus, and Crocker refers to a similar case. Xanthoma multiplex in children is not associated with jaundice, and may be congenital. In some of the cases in children there has apparently been an hereditary tendency.

Histology.— The lesions of xanthoma are growths of connective tissue which undergo partial or complete fatty degeneration. The connective-tissue increase is, in the opinion of most authorities, the result of an inflammatory process, probably of toxic origin, although, according to Touton and others, the process is essentially a noninflammatory connective-tissue hyperplasia with subsequent fatty degeneration. The fat cells are embraced in the fine meshwork of connective tissue.

In xanthoma planum the lesions are situated in the deeper part of the corium, the papillary layer being unaffected. The nodular lesions of xanthoma multiplex are of the same character as the lesions of xanthoma planum. They contain, however, a much firmer stroma of connective tissue, and are more superficially located in the corium, involving the papillary layer as well as the deeper parts of the corium.

The epidermis in both forms of xanthoma is unaffected except for a deposit of yellow pigment granules in the rete cells and in a varying degree of vacuolation of the cells of the rete.

According to Pollitzer, the lesions of xanthoma tuberosum are connective-tissue neoplasms which undergo fatty degeneration, but he believes that the lesions of xanthoma planum are the result of fatty degeneration of misplaced embryonic muscle fibers. This view has not gained wide acceptance.

Diagnosis.—The yellow, soft lesions of xanthoma placed under the epidermis are characteristic, and can hardly be confused with anything else. The lesions about the eyelids may be confused with milium, which occur also about the eyelids; milium, however, is an accumulation of sebaceous matter in the skin. The lesions are white, and if they are punctured their contents can be readily shelled out. Xanthoma multiplex has been confused with urticaria pigmentosa. In urticaria pigentosa, however, the wheallike character of the lesions, if not manifest, an readily be elicited by rubbing.

Prognosis and Treatment.—There is practically no prospect of anthoma disappearing of itself, and treatment is only successful when he lesions are few. The only way to remove xanthoma lesions is to estroy them, either by mechanical or chemical means. Individual esions may be destroyed by the applications of caustics, such as trihloracetic acid. If a caustic is used, it should be carefully limited o the surface of the lesion. For the removal of a large number of esions probably the best application is twenty-five-per-cent salicylic-acid plaster, as suggested by Morrow for the treatment of palmar and plantar anthoma. This plaster should be worn continuously for several days, or until the surfaces become irritated and the lesions softened. Another application, better adapted to individual lesions, is salicylic acid 1 dram to flexible collodion 1 ounce.

A quicker and more satisfactory way to destroy the lesions is by electrolysis, used in the same way as in destroying other small growths, as verrucæ. Probably a very efficient and convenient method of destroying these lesions would be to freeze them deeply by the application, under pressure for ten or fifteen seconds, of liquid air or solid carbon dioxide.

III.—XANTHOMA DIABETICORUM[1]

Xanthoma diabeticorum is a form of xanthoma occurring nearly always in association with diabetes and characterized by an eruption of xanthoma lesions which arise from inflammatory papules.

Symptomatology.—The eruption of xanthoma diabeticorum begins as inflammatory papules of dull red color. In all or part of these the fatty degeneration of xanthoma occurs in the tops of the lesions, thus forming papules with inflammatory bases, upon which are situated tips of chamois-yellow xanthomatous tissue. The lesions thus differ chiefly from those of ordinary xanthoma in being of frankly inflammatory haracter. The degeneration does not occur in all of the papules, so hat many of them may remain without the yellow tops. The change ecomes more uniform in the more marked cases, and in these the xanthomatous tops may be found on all of the papules. The papules vary from the size of a pinhead to that of a pea. They may be conical, rounded, or in the largest lesions flattened. They are firm to the touch, with dull red bases, around which dilated capillaries may be present, and when they have the characteristic yellow tops they may look very much like inflammatory pustules. As a rule, the eruption is scant, but in some cases it is abundant and widely generalized. The lesions are usually discrete, but upon sites where they are most abundant they may become confluent into larger tumors. The commonest locations are the buttocks, the elbows, and the knees. They may also occur on the trunk, the extensor surfaces of the limbs, the face and scalp, and rarely in the mouth. The flexures usually escape. As a rule, they avoid the eyelids, but in cases of Besnier and Hardaway they were found in this common location of xanthoma planum. The eruption appears rather suddenly, usually upon the extensor surfaces of the forearms, and then spreads to the other areas of predilection. The eruption, when once established, may remain stationary for from a few months to several years—five to seven in the longest cases—but the tendency is ultimately spontaneously to disappear. In some of the cases, notably Johnston's, the extent of the eruption has varied with glycosuria, decreasing or

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[1] *Trans. London Patholog. Soc.*, 1883, vol. xxxvi, p. 278.—Johnston, *Jour. Cutan. Dis.*, 1895, p. 401 (review and bibliography).—Robinson, *Trans. Amer. Derm. Assoc.*, 1896.—Walker, *Brit. Jour. Derm.*, 1897.—Schwenter-Trachsler, *Monatshefte*, 1898, vol. xxvii, p. 209 (complete review).—Sherwell, *Jour. Cutan. Dis.*, 1900.

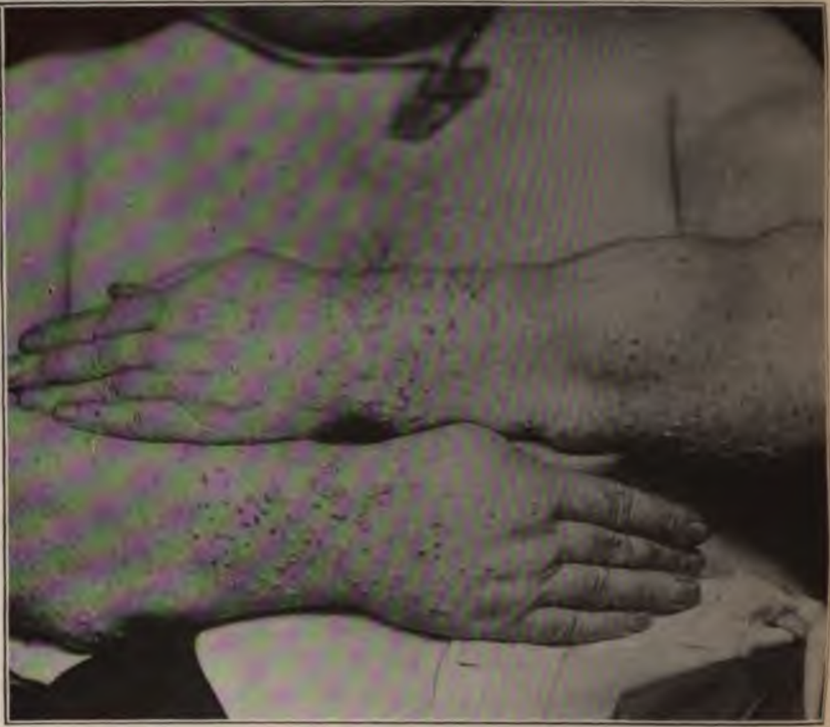

FIG. 263.—XANTHOMA DIABETICORUM, WITH MULTIPLE LIPOMATA
(Author's collection.)

icreasing as this became better or worse. The eruption may entirely isappear and recur. Upon disappearing the lesions leave no trace.

Unlike other forms of xanthoma, xanthoma diabeticorum is usually ccompanied by itching, burning, or tenderness; the difference is explainable by the more acute inflammatory type of the lesions.

Etiology and Pathology.—The condition is very rare, less than rty cases having been reported. Ninety per cent of the patients have en males; all cases except Pollitzer's—an atypical case in a boy seven-en years old—have been adults from twenty-one to fifty-seven years old.

In the great majority of cases, but not invariably, the disease is sociated with diabetes mellitus. In a few cases this has been lacking. n Colombini's case there was pentosuria. In a few cases there has en albuminuria without glycosuria. Jaundice has only exceptionally en present.

The patients have generally been rather vigorous adult diabetics, and sually where glycosuria has not been present the patients have been f the obese type who are subject to diabetes. In a few cases glycosuria as developed after the eruption, so that the eruption may be regarded n some cases as an early evidence of the tendency and as of diagnostic portance in the determination of that condition.

Anatomically, the lesions are closely similar to those of xanthoma iplex, except that there is a much more active and inflammatory cess and much less connective-tissue increase. The association of lycosuria so frequently with xanthoma diabeticorum indicates strongly at the eruption is of toxic origin, and the fact is an additional argu-ent in favor of the view that xanthoma multiplex is a toxic eruption.

The relationship of xanthoma diabeticorum to xanthoma multiplex s a moot question. Clinically and histologically, the eruption bears he characteristics of xanthoma multiplex of a distinctly inflammatory ype, a characteristic which is only one of degree and not an essential ifference. There have also been some borderland cases, especially ollitzer's in a boy of seventeen who, while he had diabetes, had a anthoma which approached more closely the ordinary type than the iabetic type. I, on the other hand, have had a xanthoma multiplex f extraordinary extent in a boy who showed the most extreme type of iabetes insipidus. The eruption in this case showed no macroscopic vidences of inflammation, but it varied in intensity with the intensity f the polyuria, as some of the cases of xanthoma diabeticorum have aried with the intensity of the glycosuria. With these transitional ases, and with the anatomical and clinical resemblances of all forms f generalized xanthoma, there seems to be no good reason to regard anthoma diabeticorum as an affection entirely apart from other forms f generalized xanthoma.

Diagnosis.—The disease can readily be confused only with simple xanthoma multiplex. The most characteristic difference from that eruption is in the inflammatory character and the red color of the bases of the lesions of xanthoma diabeticorum. The lesions begin as red papules, and the typical xanthoma tissue may be absent from some of them. The evolution is rapid; there is a distinct tendency to involution, and there are usually tenderness and itching, all features distinguishing the condition from xanthoma multiplex. Xanthoma diabeticorum is associated with glycosuria and not with jaundice.

Prognosis and Treatment.—The eruption tends to disappear spontaneously sooner or later—in from a few months to several years. Its disappearance can be greatly hastened by treatment of the underlying disturbances.

Its treatment is directed to the treatment of diabetes where diabetes exists, or to the removal of a presumed diabetic tendency in cases where actual glycosuria is not present. Crocker recommends arsenic internally on theoretical grounds. For the relief of the subjective symptoms a simple antipruritic application, like calamine lotion, or one-half- to one-per-cent menthol ointment, are sufficient.

IV.—COLLOID DEGENERATION OF THE SKIN[1]

SYNONYMS: *Colloid milium* (Wagner), *Hyaloma.*

Colloid degeneration of the skin occurs in the form of a scant eruption of semitranslucent, yellow, deeply embedded papules, usually located upon the face.

It is an excessively rare affection, which was first described by Wagner in 1866.

Symptomatology.—The lesions are pinhead- to small pea-sized, flattened papules which, while slightly elevated, are deeply embedded in the skin. They are yellowish to lemon yellow, and shiny and translucent, so that they look like vesicles, but upon opening them only a jellylike mass or a drop of blood can be pressed out. They may have a reddish areola, showing dilated capillaries. They are soft and elastic to the touch.

The usual location of the eruption is the upper two thirds of the face—the cheeks, the bridge of the nose, the forehead, and around the eyes; in Perrin's case and in C. J. White's case they occurred also on the backs of the hands, and in Perrin's case on the conjunctivæ. The lesions occur in groups of two or three, and gradually increase in number. Adjacent lesions may apparently coalesce to form larger

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[1] Perrin and Reboul, *Trans. Second Internat. Cong.*, 1892.—C. J. White, *Jour. Cut. Dis.*, 1902, p. 49 (review of recorded cases and histology).

but the individual lesions even then remain distinct. The
when once established, persist, as a rule, though individual
ave been observed to undergo spontaneous involution, beginning
nter of the lesion. In Liveing's case the eruption disappeared
ously, some of the growths disappearing by inflammation, fol-
crusting, and leaving very faint scars. The eruption is without
e symptoms.

logy and Pathology.—Only seven undoubted cases of the dis-
recorded. It has occurred in both men and women at various
r sixteen years. There is no definite association with any other
except that in two cases there has been preceding or accom-
neuralgias or headaches. In some of the cases habitual exposure
eather, especially to sunlight, has apparently been the exciting

disease is not, as Wagner thought upon clinical grounds, a
tion of the sebaceous glands. The studies of Besnier and
C. J. White, and
ave demonstrated
s a colloid degene-
of the connective
f the corium, in-
that of the walls
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tion begins in the
art of the corium,
rly around the se-
glands and hair
White in his case
hat in the lesions
llæ were obliterat-
the corium was
d into a homogene-
s of colloid mate-
rounded by a thin
elacin. "Within
e of elacin we find
or proper consist-
small or large, ir-
shaped islands of tissue, bounded at intervals by capillaries
alls show thickened masses of elacin. . . . Within these inclos-
llary walls the true colloid material appears composed of a
ork of fine or coarse granules, which stain uniformly yellow
ric acid. . . . Scattered through this homogeneous mass, one

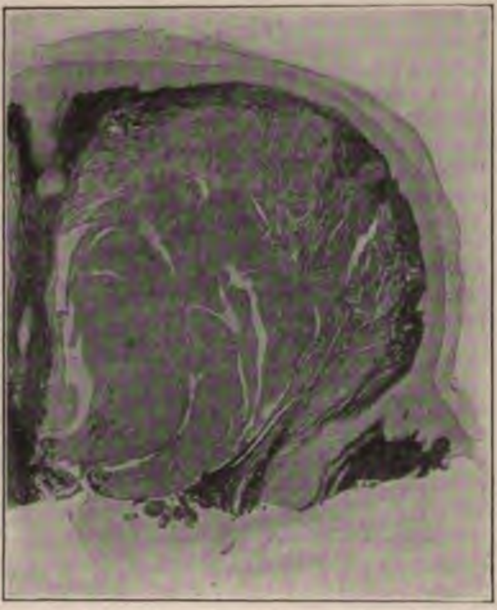

FIG. 264.—COLLOID DEGENERATION OF THE SKIN. (C. J. White's collection.)

is surprised to note perfectly preserved connective-tissue nuclei and, on approaching the encapsulating vessels, to find many surrounding leucocytes with a rare plasma cell."

Philipson has tried to show that hydradenoma, benign cystic epithelioma, and colloid degeneration of the skin are the same, but neither the clinical pictures nor the histological findings bear out this contention.

Diagnosis.—The condition must be distinguished from milium, xanthoma, hydrocystoma, and benign cystic epithelioma. In most cases the characteristic clinical features of these various affections would readily differentiate them. In any case, microscopical examination would be necessary fully to establish the diagnosis of colloid degeneration of the skin.

Treatment is without effect except in so far as the lesions are individually destroyed by curetting, electrolysis, or other similar means.

Colloid Degeneration in Granulation and Scar Tissue

Juliusberg[1] has described three cases of colloid degeneration occurring in granulation and scar tissue which correspond to his title given above. The disease appeared in patches varying in size up to an inch or more in diameter. The patches were yellowish or yellowish brown, and were made up of isolated and confluent light yellow, slightly elevated plaques having a translucent appearance. The patches were not due to pigment deposit, or to collections of cells as in xanthoma, but to a peculiar degeneration of the elastic and fibrous tissue staining like colloid material, and Juliusberg groups the affection with colloid milium. In two of the three cases the process occurred in granulation tissue; in one it occurred in scar tissue.

Degeneration of Elastic Tissue[2]

Under the title "A New Form of Circumscribed Degeneration of the Skin," Dreuw cites the case of a man fifty years old who for a year had had a recurrent pustular eruption about the malleoli of the right leg. The eruption consisted of confluent crusts and scabs on an erythematous and swollen base; removal of the scabs left serpiginous ulcers, which were covered with a seropurulent exudate. Clinically, the eruption showed nothing peculiar except the recurrences, which were probably produced by the patient. Beneath the epidermis there were visible peculiar yellow bodies the size of a poppy seed, which persisted during the intervals between the recurrent ulcers; these are the lesions to which Dreuw especially refers, the ulceration being accidental, and probably an artificially produced complication.

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[1] *Archiv*, 1902, vol. lxi, Nos. 2 and 3.

[2] *Monatshefte*, vol. xxxvi, 1903, p. 629.

Histologically, these bodies were composed of longitudinally and transversely running bundles of parallel fibers which were highly refractile and very brittle, resembling vegetable rather than animal tissue. These fibers took an atypical orcein stain, and Dreuw thinks they are elastic fibers which have undergone a peculiar and hitherto undescribed form of degeneration. Normal elastic fibers, in fact, could be seen to become degenerated as they entered the area.

Over the area of degeneration the papillæ were almost obliterated, and there was a marked hypertrophy of the rete malphigii, the upper layers of which were œdematous and stained poorly.

V.—DERMOLYSIS[1]

A condition to which he has given this title has been described by C. J. White upon the study of one case in a baker, aged twenty-five. Lesions occurred about the elbows and on the thighs above the knees. The early lesions consisted of dome-shaped, cherry-colored papules of he size of a pea, which were firm, tense, and movable. These papules very slowly developed into muddy-white, flattened, finger-nail-sized papules. Lesions either occurred isolated or were strikingly grouped around a relatively depressed, bluish-red center. The disease had existed for several years, and the evolution and involution of lesions were very slow. There were no subjective disturbances. Histologically, there

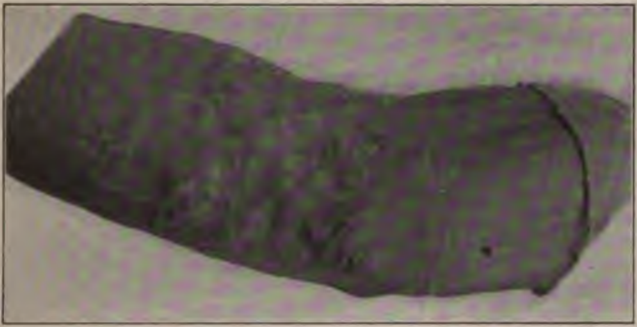

FIG. 265.—MACROSCOPIC LESIONS OF DERMOLYSIS.
(C. J. White's illustration.)

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[1] C. J. White, *Jour. Cutan. Dis.*, July, 1908.

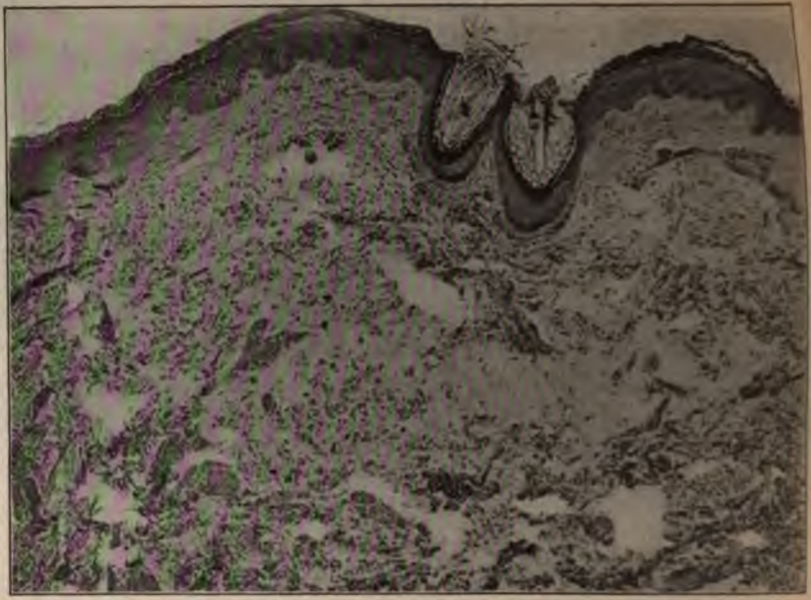


FIG. 265*a*.—DERMOLYSIS. A DOME-SHAPED, CHERRY-COLORED PAPULE. Note the apparent absence of nuclei in the rete; the real absence of granular cells; the focal disintegration of the corium with the relative persistence of cellular elements. × 75. (C. J. White's illustration and description.)

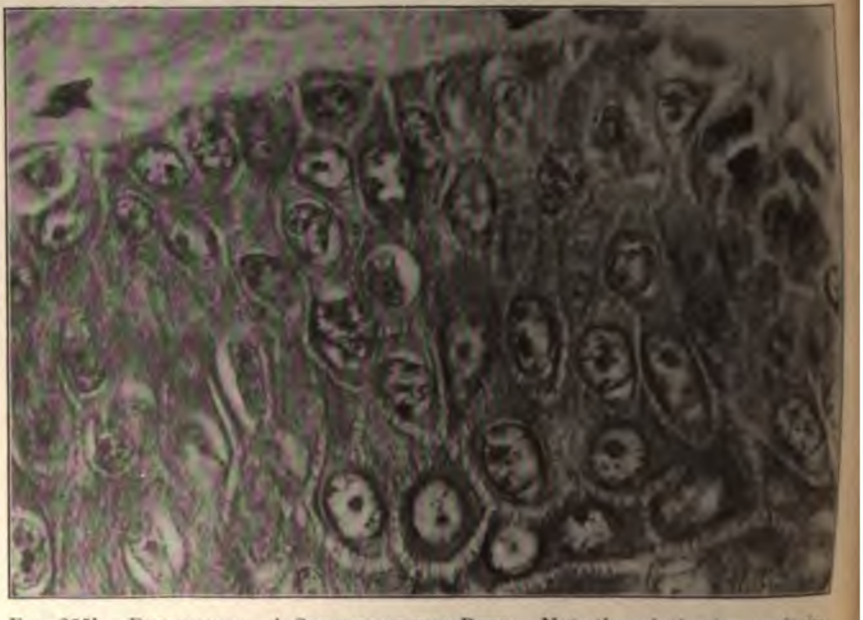


FIG. 265*b*.—DERMOLYSIS. A SECTION OF THE RETE. Note the relative increase in size of all the nuclei; the decrease in protoplasm of most of the nuclei; the distortion of some of them; and the total disappearance of others, with final loss of their cell boundaries. × 1,500. (C. J. White's illustration and description.)

was a remarkable rarefaction of the skin. The epidermis showed degenerative changes; the demarcation between the epidermis and corium in some places was well marked, in others ill defined. The papillæ were flattened. The essential lesion was in the corium, from the subpapillary layer downward. It was not a degeneration into a colloid mass, but an almost complete disintegration of the tissue elements of the corium.

The disease dated back to a time when the boy had slept under conditions exposing him to an unusual degree of heat, and White regards this exposure to heat at that time and subsequently in his occupation as a baker as probably the factor of greatest importance in the production of the disease.

B. BENIGN NEOPLASMATA

I.—CICATRIX

SYNONYM: *Scar.*

A scar is a connective-tissue formation covered by epidermis, which is the result of a reparative inflammatory process and replaces a previous loss of substance of the corium.

Scars may follow not only wounds, but diseased processes of the corium, like lupus erythematosus, which are characterized by loss of substance without ulceration. Usually, however, a scar represents a previous wound of the corium. In wounds which involve only the epidermis, normal epidermis forms over the surface without the production of scars.

Scars are divided into traumatic and pathological scars, depending upon whether they are the result of traumatism or of disease; but the division is artificial, and of no practical importance.

A scar is formed by the development of granulation tissue, which later becomes fibrous. A newly formed scar is soft, and pink to reddish. After its fibrous structure becomes established and the inflammatory process disappears, the scar becomes whitish and of firm texture. More or less redness may at times persist as the result of an abundant capillary network, and there may also remain pigmentation which is usually hematogenous. Except in dependent positions where the circulation is poor, this deposit usually is absorbed. With the conversion of the granulation tissue of the scar into fibrous tissue, more or less contraction takes place. In linear scars this simply serves to bind together the parts, but in scars of large extent the contraction frequently produces much distortion of the surrounding structures.

The character of a scar depends upon the extent of the loss of substance which it must replace, and upon the thickness of scar tissue which is produced. After an aseptic linear wound, where there has been a minimum reaction, the scar is usually a simple line, neither elevated nor depressed. Where scar tissue must replace a considerable loss of substance in the skin, as in an ulcer, the scar tissue may not be sufficient to completely fill the previous wound; in such a case there may result a depressed scar which, depending upon its thickness, contracts more or less. Frequently, as a result of infection or of other factors involving the formation of granulation tissue, there is an excessive amount of scar tissue formed, resulting in a hypertrophic scar. Such hypertrophic scars, which have been especially described by Dieberg, present no essential difference from ordinary scars, but are simply excessive in amount. Hypertrophic scars are usually the result of infected wounds or wounds of considerable extent in which, in order to obtain healing, a prolonged process of granulation-tissue formation is necessary. The size, shape, and other features which characterize scars depend upon factors in the disease processes which cause them. As has been seen in the consideration of various diseases in which scarring occurs, these features are often of important diagnostic significance.

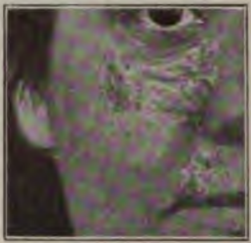


FIG. 266.—HYPERTROPHIC SCAR. (Author's collection.)

Anatomically, a scar ordinarily presents a plain connective-tissue surface covered by epithelium. Usually the papillary surface of the corium is entirely obliterated, but, according to Heitzmann, papillæ may persist in scars. The epidermis over scars is usually thin, but it may be hypertrophic; otherwise its structure is normal.

The treatment of scars is the same as that of keloid.

II.—KELOID

SYNONYMS: *Cheloid, Alibert's Keloid, Kelis, Kelos, Cheloïde* (Fr.), *Keloide.*

A keloid is a connective-tissue new formation essentially identical with a hypertrophic scar, and occurring in most, if not in all, cases after traumatism. Leaving out of consideration for the moment the finer distinctions which are drawn between keloids and scars, a keloid may be regarded as an exuberant scar.

Keloid was first described as such by Alibert.

Symptomatology.—Keloids are divided into false keloids, those originating from traumatisms or wounds, and true keloids, which are supposed to develop spontaneously. There is no way to distinguish en these two kinds except by the history, and it is doubtful if ever originates without a previous traumatism.

eloids in all of their features correspond to excessive scars. Doubt- ecause they originate most frequently in linear wounds, most s are elongat- spindle-shaped, hey may have rregular forms. frequently there idges running om the body of eloid, buttress- s it were, and ting a fanciful blance to claws, which the name erived—χηλή, a Keloids may ered with skin tly normal in arance. Fre- ly the surface is tense and glistening; it may be dead white or ish, and around and over it there may be dilated capillaries. As a rule, keloids are not numerous. Usually there are only one or two or a few, but in some cases their number may be hundreds. They may occur at any point; their commonest location is on the upper part of the chest, especially over the sternum and breasts of women. When numerous they are apt to be symmetrical in distribution, doubtless because of the symmetry of the previous lesions from which they develop.

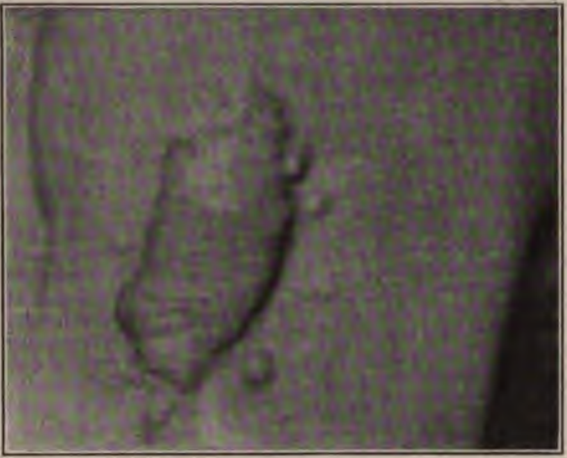
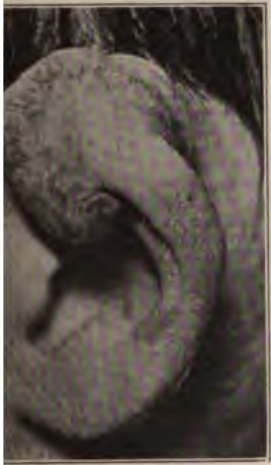


FIG. 267.—KELOID. (Author's collection.)



IG. 267*a*.—KELOID. Author's collection.)

In rare cases keloids occur as circumscribed, hard plates embedded in the skin, and elevated very little or not at all above the surface. This so-called keloid *en plaque* of Hutchinson and R. W. Taylor is regarded by Unna as a fibroma. Keloids grow slowly, requiring months eral years to reach a fixed size. After a certain length of time usually become stationary. In rare cases they increase indefinitely

—from ten to twenty or forty years—and in such cases the tumors may reach large size. In some cases, but not frequently, partial or complete involution occurs; its course, as a rule, requires from one to two years or longer, but complete involution has been observed in a few months. In a unique case observed by Erasmus Wilson, the keloids varied in size with the state of the patient's health, increasing when the health was poor. In very rare instances malignant degeneration has been observed in keloids as in other scars. In many keloids there are no subjective symptoms, but in many, I should say in the majority, there is increased tenderness, and in some there is itching or spontaneous pain. In rare cases the pain is so acute as to constitute an important symptom requiring relief.

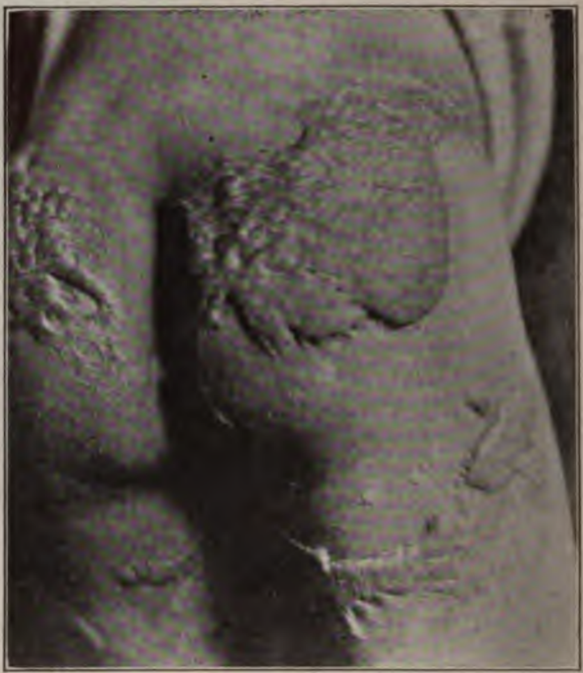


FIG. 267*b*.—KELOID. (Grover W. Wende's collection.)

ACNE KELOID is a mass of hypertrophic scars which occurs on the nucha, at the border of the scalp, as a result of a suppurative folliculitis, known as *dermatitis papillaris capillitii.*

Other forms of keloid, which have been described but which present no essential peculiarities, are the *verrucose cicatricial tumors* of Hawkins and the *syphilitic keloid* of Wilkes.

Etiology.—The reason for the occurrence of keloids is unknown. They occur most frequently in negroes, in whom there is a peculiar tendency to fibrous-tissue hyperplasia of all sorts, and there is often marked individual predisposition to their development. Frequently, also, there is a manifest family and hereditary tendency. They occur at all ages and in both sexes. They have been seen in earliest infancy, and one case of congenital keloid has been observed by Bryant; but they are seen most frequently in adults, and are commoner in women.

Keloids may result from wounds of any size—from such minute wounds as those arising from insect bites, or the piercing of the skin by a needle, or from all kinds of inflammatory lesions of the skin like those of acne or smallpox—and Crocker records an extensive case which followed so slight an inflammatory process as prickly heat. I have seen one severe case of multiple keloids upon the chin following electrolysis for the removal of hair. They may result not only from dermatoses accompanied by abraded surfaces or suppuration, but they have been noted occasionally in association with or subsequent to morphea, fibromata, lipomata, and psoriasis. It must also be remembered that they may result from traumatism, such as results from blows, pressure, or friction, without any solution of continuity. They are most likely to follow suppurating wounds, but suppuration is by no means necessary for their occurrence. It is evident from the slight injuries which may at times result in keloids that it is excessively difficult to eliminate the possibility of traumatism in what are apparently spontaneous keloids, and it is on the whole highly probable that spontaneous keloids never occur. This was in substance the opinion of the committee of the London Clinical Society which was appointed for the consideration of keloids.

Pathology.—The only distinction that will hold between a keloid and a hypertrophic scar is that the keloid extends beyond the limits of the original scar. This is hardly a vital distinction. The inflammatory process which results in the production of scar tissue is not confined to the limits of the wound which it is designed to replace, nor is the scar tissue itself, and it does not indicate any departure from the essential character of a scar if in keloids it far exceeds these same limits. Histologically, a keloid consists of bundles of dense homogeneous connective-tissue fibers running, as a general rule, parallel to the long axis of the growth, but crossed here and there by transverse fibers. Between these bundles are seen round and spindle-shaped connective-tissue cells, these being especially numerous about the blood vessels, and even extending into the periphery. The growths commence in the corium and grow to the surface, being separated from the rete by a loose vascular layer of connective tissue, the compressed corium. A keloid is not sharply limited from the surrounding tissue, but in places sends out prolongations into the healthy tissue. The appendages of the skin are lacking in a keloid, having been pushed aside. In those keloids originating in scars, the papillæ are usually absent; but in the so-called spontaneous form they are present, but always somewhat deformed by pressure.

Diagnosis.—The technical distinction made between a scar and a keloid is that the keloid extends beyond the limits of the original scar. It may also persistently grow for many years, and be spontaneously painful. These characteristics are sufficient to distinguish the clinical picture of well-defined keloids from ordinary hypertrophic scars. Occasionally keloids occur in the form of globular tumors, which closely resemble sarcomata. Such lesions, unless associated with other typical keloids, might be distinguished only with extreme difficulty from sarcomata; usually, however, the association with typical keloids and the very slow growth would readily indicate the character of the lesions.

Treatment.—The treatment of keloids has hitherto been very unsatisfactory. In recent years various practitioners, including myself, have been able to convert them permanently into flat, flexible white scars by the application of x-rays. My experience covers a good many cases of hypertrophic scars, and several cases of typical keloid; in none have I failed to get either great permanent improvement or complete flattening of the scar. At times this result may be obtained without producing any apparent reaction in the tissues. Occasionally it is necessary to carry the exposures to a point of producing a moderate dermatitis. The exposure should be given cautiously from day to day, or two or three times a week, depending upon the intensity of each exposure, and gradually carried to the point of producing an erythema unless the growth subsides before this develops. The time required for treatment, in my experience, varies from one or two to five or six months. Where the treatment extends over the longer periods it is because the series of exposures have to be repeated several times.

Various other plans of treatment are used: multiple deep scarification (Vidal), the injection into the tumor of a few drops of twenty-per-cent solution of creosote in oil (Marie), the injection around the keloid or scar of ten to twenty drops of thiosinamin in alcohol, or, better, in equal parts of water and glycerine (Tousey, Newton, Van Hoorn), electrolysis (Hardaway). These methods are all unreliable. Those most likely to be followed by improvement are electrolysis and the injection of thiosinamin, followed by massage. This latter method usually causes marked improvement, but recurrence takes place. Administration of thyroid extract has in a few cases been followed by improvement. Some improvement may be obtained by the continuous application of mercurial plaster or lead plaster. Excision of keloids is not a good procedure. It is almost invariably followed by the reappearance of a larger tumor.

III.—FIBROMA

Fibromata of the skin occur in the form of soft, abruptly elevated, either sessile or pedunculated tumors which are of the consistence of soft connective tissue.

Small connective-tissue tumors are not infrequent in the skin. They may occur as embedded tubercles of normal or pinkish color, from the size of a small to a large pea. These occur singly, or there may be several over the body surface. They are usually discovered accidentally, show no tendency to increase in size, and are of no importance. In old age there are not infrequently found one or more pendulous, teatlike fibromata, especially on the face, neck, and back. They are of normal color, and they likewise cause no inconvenience and are of no practical importance.

FIBROMA MOLLUSCUM[1]

In addition to these wholly unimportant lesions there is a well-defined clinical entity in which there occur one or more large, or many small, abruptly projecting connective-tissue tumors. This condition is variously known as *fibroma molluscum, molluscum simplex, molluscum pendulum,* and *neurofibroma,* or von Recklinghausen's disease. It was regarded as a pure fibroma until von Recklinghausen demonstrated that the lesions, at least in some instances, correspond to neurofibroma.

Fibroma molluscum occurs in three clinical varieties: first, as multiple fibromata, usually of small size; second, pendulous fibromata, in which there are one or a few large, pendulous tumors; third, as a mixed form, in which both large, pendulous tumors and small, multiple tumors are present. These three forms are all varieties of one pathological entity.

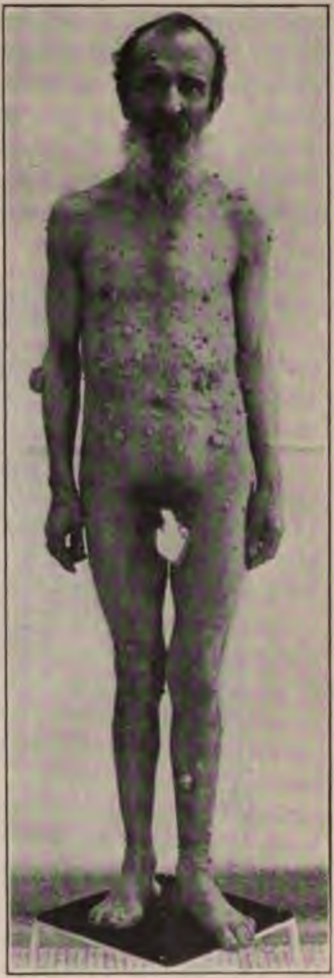


FIG. 268.—MULTIPLE FIBROMA. (Author's collection.)

Multiple Fibroma.[2]—Multiple fibromata occur in the form of many or innumerable connective-tissue tumors scattered over the body. The tumors are of all sizes up to an orange or larger. They project abruptly above the surface, and are either sessile or pedunculated. They may be round or teat-shaped, or slightly pendulous, and are of the consistence of soft connective tissue. Usually their color is that of normal skin. They may be pink from increased vascularity, and frequently they are pigmented. Anthony regards the pigmentation as a primary process, which may be present even before the appearance of the tumors, and lays particular emphasis upon its occurrence. The number of these tumors may literally be almost beyond enumeration, and as the tumors are prominent they produce a most striking and characteristic clinical picture. They occur most abundantly on the trunk, but are also found on other surfaces. They are rare in the palms and soles, and on these sites are usually flattened from pressure. Occasionally they occur upon the tongue and other surfaces within the mouth. The affection develops slowly both in size and number of lesions. After a certain length of time the condition tends to remain fixed, but involution of the tumors may occur. Upon disappearance of the connective-tissue hyperplasia, bladderlike lesions of the skin may be left which are probably exactly

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[1] Hartzell, *Am. Jour. Med. Sci.*, February, 1902.

[2] Anthony, *Jour. A. M. A.*, 1903, vol. xl, p. 1630.

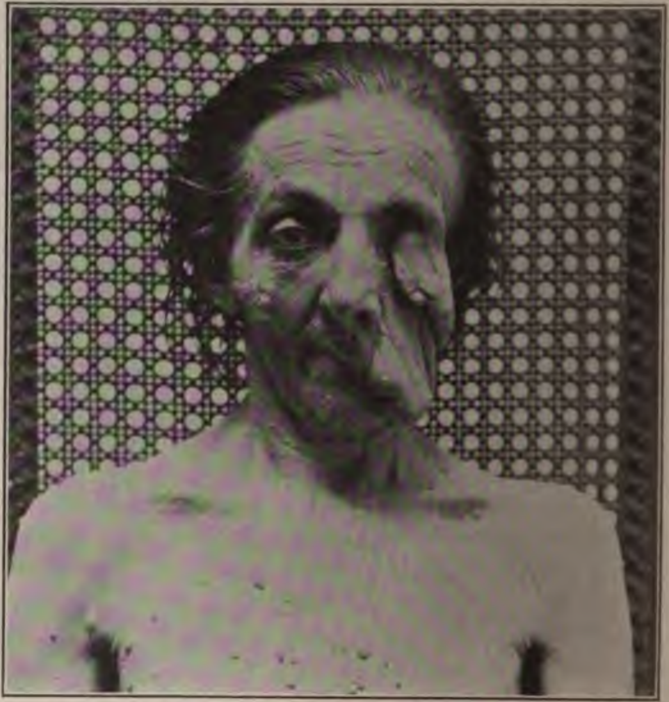

FIG. 269.—MULTIPLE FIBROMA WITH FIBROMA PENDULUM.
(F. G. Harris's photograph.)

f the same character as the lesions described as multiple benign tumor-ike growths. The skin over the lesions, except for the pigmentation, s usually normal. Hairs are most frequently lacking on the tumors, nd the surface of the skin is smooth, but occasionally there are one r more greatly dilated sebaceous glands.

Fibroma pendulum occurs in the form of large, pendulous tumors, hich are usually single. They vary in size from a small pear, to which hey can be accurately compared in shape, up to enormous masses larger han a head, which produce great disfigurement or great mechanical nconvenience. The skin over them usually shows dilated, plugged ebaceous follicles, and is frequently pigmented. Sites of predilection or these lesions are the face and neck, but they occur in various locaions. From the drag which they cause upon other tissues they may roduce serious distortion of the parts.

Both multiple fibroma and pendulous fibroma are without subjective ymptoms, and cause no annoyance except such as arises from the disigurement or the mechanical disturbance.

Fibroma molluscum gravidarum.—Under the above title, Brickner[1] has called attention to the appearance of fibroma molluscum during regnancy, and, although it is a hitherto undescribed clinical entity, he as seen nine cases of the condition.

The tumors appeared from the fourth to the sixth months of pregnancy, and were small, pigmented or nonpigmented, sessile or pedunulated growths. The growths were from ten to fifty in number, and aried in size from a pinhead to half of a split pea. They were most ommon in the upper half of the body, especially about the neck. Brickner has not seen them on the extremities or the mucous membranes. They increased in number as pregnancy advanced, and disappeared hortly after parturition. No causative features beyond pregnancy were discoverable. Brickner thought of the possibility of disturbance of the hyroid gland during pregnancy as of etiological importance, but found no symptoms to support it. Histologically the tumors were connective-issue growths involving the papillary layer, with numerous connective-issue cells and blood vessels, the general appearance being that of a "soft fibroma moderately rich in connective-tissue cells." No muscle r nerve fibers could be found, although the tumors were examined for erve tissue with especial care. The epidermis was normal, and the asal layer pigmented, the pigmentation fading out at the edge of the umors.

Etiology and Pathology.—Fibroma multiplex usually begins in arly life, and in some cases has been congenital. The cases, however,

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[1] Brickner, *Amer. Jour. Obstet.*, vol. liii, No. 2, 1906.

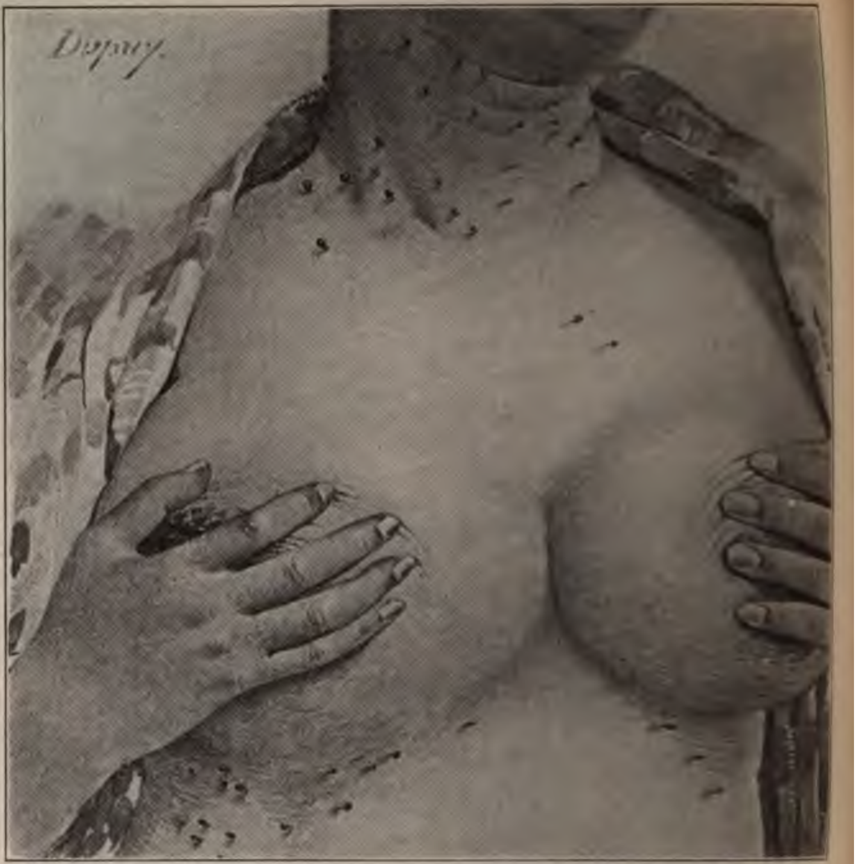

FIG. 270.—MOLLUSCUM GRAVIDARUM. GROSS APPEARANCE. (Brickner.)

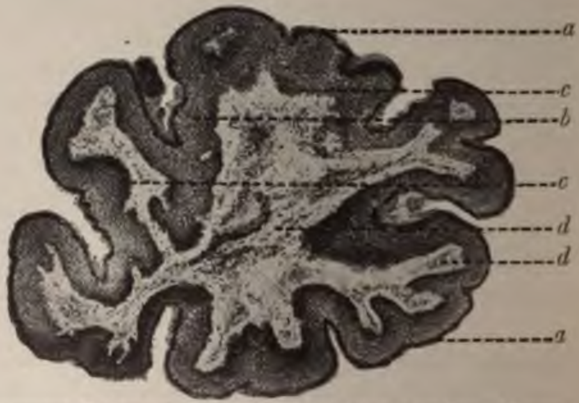

FIG. 270*a*.—MOLLUSCUM GRAVIDARUM. PHOTOMICROGRAPH OF CROSS-SECTION. *a*, Normal skin; *b*, hypertrophied papillary layer; *c*, deposits of pigment; *d*, young connective tissue. (Brickner.)

reach their full development only in later life. Fibroma pendulum is usually acquired in later life. Fibromata occur in both sexes, but are apparently more common in males. They are perhaps commoner in the darker races. In many of the cases there has been a marked hereditary tendency. According to Hebra the disease is usually associated with stunted mental development, but this is by no means the universal observation.

On account of the presumed mental deficiency of the patients a relationship to myxœdema has been suggested, but the facts do not bear it out. The highly interesting group of cases of Brickner demonstrate the possibility of production of fibromata by systemic causes, but as to what is the nature of these we are hardly in position even to surmise.

Fibromata are produced by hyperplasia of circumscribed areas of connective tissue. They may originate probably from any of the connective-tissue structures either of the corium or of the subcutaneous tissue. The tumors are made up of wavy connective-tissue fibers containing many cells and blood vessels. In the young tumors the tissue is vascular, with many young connective cells, and may be very œdematous. The older tumors are denser and less vascular. The epidermis over the growths may be normal, or may show hypertrophy or pigmentation. Recklinghausen has shown that in multiple fibromata the tumors are often, if not always, neurofibromata, and there is a tendency now to regard these cases all as neurofibromata. It is almost inconceivable, however, that all connective-tissue tumors of the skin should spring from the connective tissue of the nerves. and that no pure fibromata occur.

Prognosis and Treatment.—The tumors, if completely removed, do not tend to recur. In multiple cases with innumerable lesions excision of all the lesions is impracticable, and the cases are usually beyond relief. However, in one case of multiple fibroma Whitehouse has caused the disappearance of the tumors by the administration of arsenic in the form of the Asiatic pill. The treatment is worthy of trial, especially in younger patients.

MULTIPLE TUMORS OF THE SKIN ASSOCIATED WITH ITCHING

Schamberg and Hirschler[1] have reported two interesting cases of multiple itching tumors of the skin in negro women. The tumors were of the consistence and appearance of fibrous-tissue growths and varied from a pea to a hazelnut in size. They were sharply circumscribed, blackish, and covered by thickened epidermis. They occurred especially on the extremities, but in one case some lesions were also on the trunk.

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[1] Schamberg and Hirschler, *Jour. Cutan. Dis.*, April, 1906.

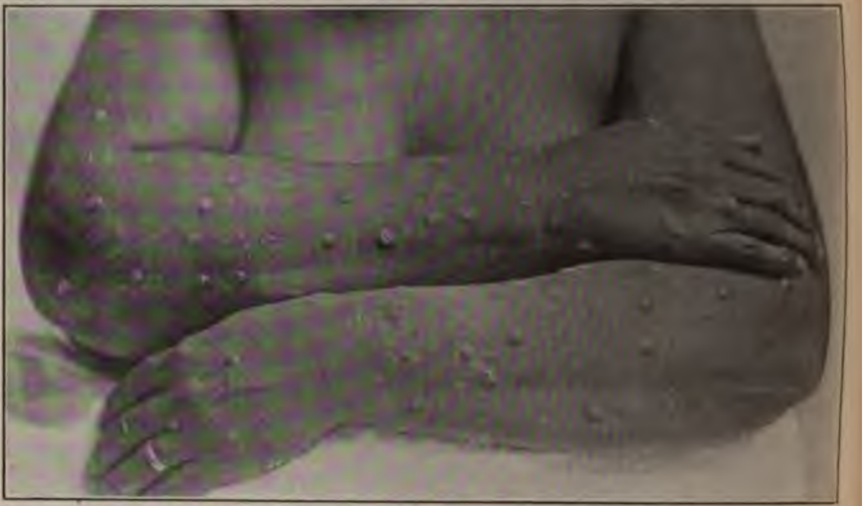

FIG. 271.—MULTIPLE TUMOR OF THE SKIN ASSOCIATED WITH ITCHING. (Schamberg and Hirschler.)

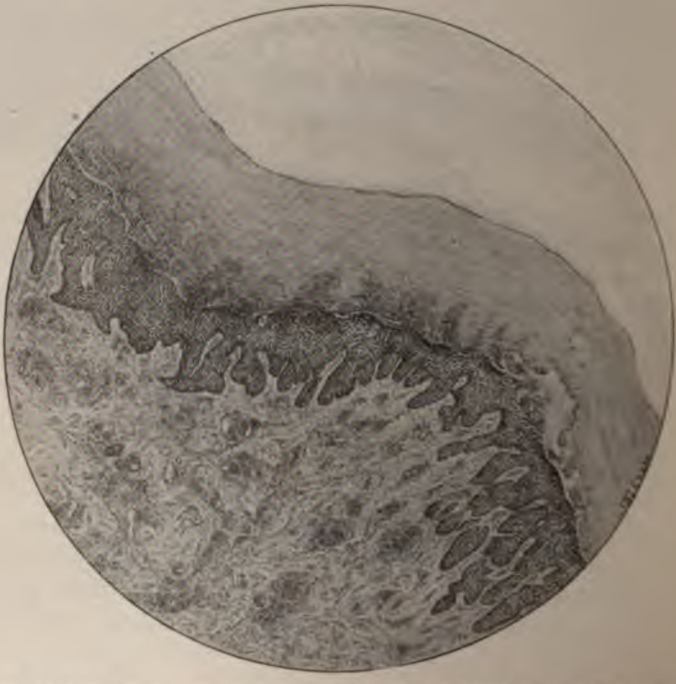

FIG. 271a.—SECTION OF TUMOR IN SCHAMBERG'S AND HIRSCHLER'S CASE.

hey were of fourteen and fifteen years' duration, recurring after re-oval.

Histologically the changes were those of a chronic inflammatory ocess with connective-tissue hyperplasia. The corium showed nests of und-cell infiltrations. The elastic tissue and collagen were normal cept for hypertrophy of the latter. Neither plasma cells nor bacteria re found. Hardaway (*Arch. of Derm.*, 1880) has described a similar se of twenty-one years' duration in a white woman.

C. J. White describes another case of this rare dermatosis. He re-rds his case, the case reported by Hardaway, and two cases reported by hamberg and Hirschler as the same condition which has been described Brocq, upon the basis of five cases, as lichen obtusus corneus. White nsiders the condition as a lichenification of an urticarial skin under ng-continued irritation. He gives a bibliography of the subject and a mplete clinical and histological study of his case.

IV.—PARAFFINOMA

Paraffinomata are tumors developing at the site of injection of par-in. Paraffin prosthesis has had a considerable vogue among cosmetic ecialists in recent years for removing wrinkles and effacing unsightly pressions about the face. It has a legitimate field of usefulness in me fields of surgery. Occasionally after these injections into the sub-taneous tissue slow-growing fibrous tumors develop as a result of the rsistent irritation from the paraffin in the tissues. These tumors are dular or ovoid, densely hard, and vary in color from normal skin-color dark violaceous. Their size, number, and arrangement vary with the nount and distribution of the paraffin in the tissues. Some of the dules develop in a few months after the injections, while, as a rule, x to eighteen months elapse before the masses begin to appear. In some the cases the nodules remain stationary after a few months' growth; others their growth steadily continues for a year or more with the roduction of tumors, up to the size, it may be, of a small egg. The tu-ors have shown no tendency to disappear. Occuring at sites where ight variations from normal are conspicuous—angles of the mouth, hin, nose, eyelids, and forehead—the tumors are very disfiguring.

These results are rare in proportion to the number of cases of paraffin prosthesis; nevertheless, the cases are coming to the attention of ermatologists and surgeons, and the disfigurements are so incomparably orse than the original defects that the possibility of the accident ren-ers unjustifiable attempts at paraffin prosthesis for trivial facial defects.

The development of these tumors is explained by the presence of an ritating foreign body in the tissues, which excites a subacute inflam-

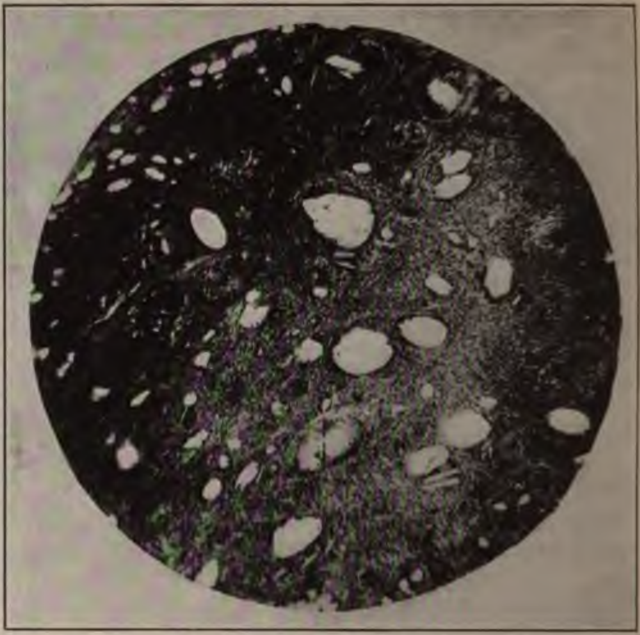

FIG. 272.—PARAFFINOMA. (Heidingsfeld's collection.)

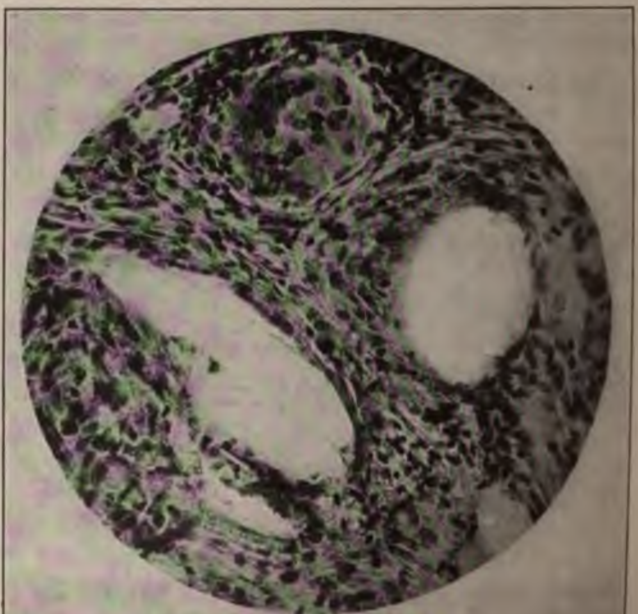

FIG. 272*a*.—PARAFFINOMA. (Heidingsfeld's collection.)

reaction with ultimate fibrosis. They are thus in their essential r like hypertrophic scars. Microscopically the tumors are masses ulation tissue, such as occur around foreign bodies, composed of onnective-tissue cells in various stages of organization, a leuco- vasion to carry off the paraffin, and numerous giant cells. A feature of the histological picture is the dilated spaces—sug- a piece of well aërated Swiss cheese (Heidensfeld)—which were ith paraffin before it was washed out with xylol in preparing the ns. The only effective treatment of the tumors is to dissect them carefully, and this is not entirely satisfactory.

V.—NEUROMA[1]

NYMS: *Neurofibroma, Nerve Tumor.*

romata are sometimes seen in the scars of amputations. Occa- spontaneous neuromata develop in the subcutaneous tissue, and essive rarity they occur in the body of the corium.

ptomatology.—Amputation neuromata are produced by the ation of the proximal end of a nerve and the involvement of it tissue. They occur as tumors up to the size of a hazelnut or which are spontaneously painful and exceedingly tender upon . As a rule, the amputation neuroma develops deep in the tis- the stump, but it may develop just below the surface as a small, ely tender tubercle. They belong to the domain of surgery.

he subcutaneous tissue, neuromata appear as hard tumors from of a pinhead to that of a pea or larger. They are rounded or shaped, usually multiple, and occur in the area of distribution rve, or of several nerves. When occurring in several branches of they constitute the plexiform neuroma. They are painful on and may be spontaneously painful.

tiple painful neuromata in the body of the corium have been l in only two cases, once by Duhring, and once by Kosinski. In g's and Kosinski's cases, the tumors were from the size of a pin- a pea, of reddish or purplish color, and attached to the skin so y were movable only with it. The lesions were sensitive to pres- d after a while were associated with severe paroxysmal pain. In g's case the skin of the entire part became hot and purplish the paroxysms of pain, but at other times the skin between the s was normal. In both cases the lesions were multiple and nu- in the affected areas. In Duhring's case the lesions occurred shoulder and arm, involving branches of the circumflex nerve;

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bring, "International Atlas," Plate XXXV.—Kosinski, *Centralblatt für* , No. 16, 1874.

in Kosinski's case, on the upper two thirds of the thigh and on the buttocks, involving the small sciatic and the external femoral cutaneous.

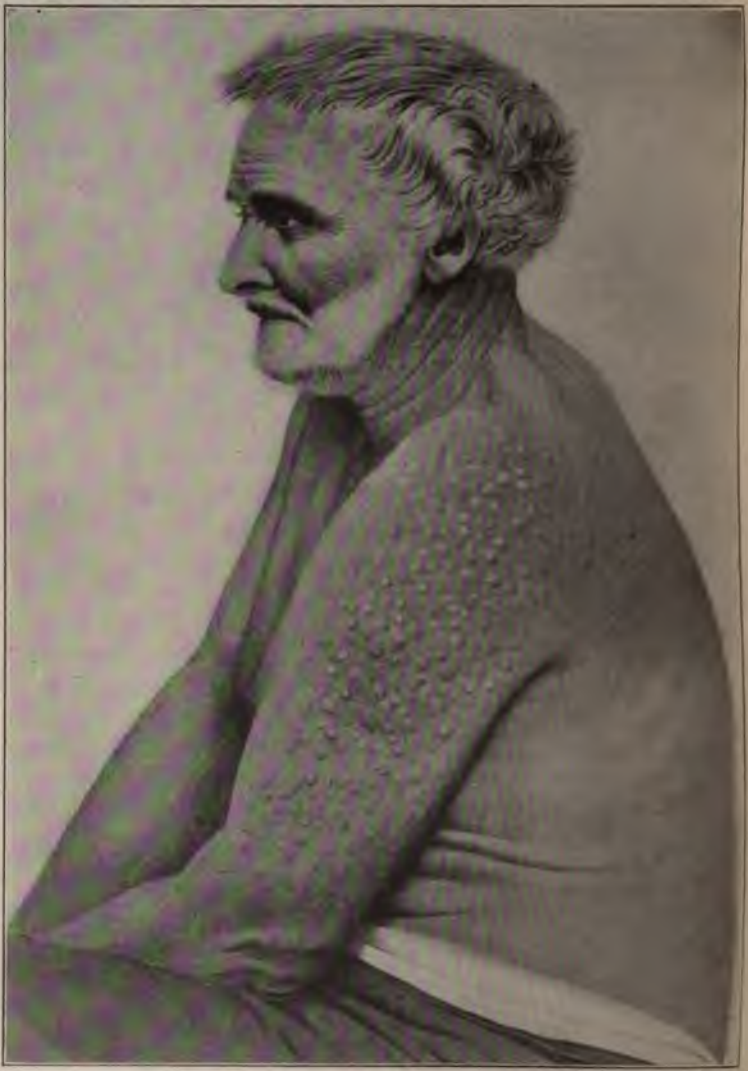

FIG. 273.—NEUROMA CUTIS DOLOROSUM.
(Photographic reproduction from Duhring's Plate, International Atlas.)

Pathology.—The pathogenesis of neuromata is not clear; they probably arise from a developmental anomaly of the connective tissue of the nerve which, on the accession of some etiological factor, such as trauma or other irritation, results in proliferation. Microscopically a neuroma onsists of a more or less dense connective-tissue hyperplasia in or over hich run either medulated or nonmedulated nerve fibers.

Treatment.—The treatment of neuromata is surgical. Single le-ions may be excised. In Duhring's case partial resection of the brachial lexus, and in Kosinski's case resection of part of the small sciatic nerve, ere followed by disappearance of the pain and subsidence of the tumors. n six months there was a recurrence in Duhring's case.

VI.—LIPOMA[1]

SYNONYMS: *Adipoma, Fatty Tumor.*

Symptomatology.—Lipomata occur in the skin both as circum-cribed and as diffuse fatty tumors. The circumscribed tumors are from ea to plum size or larger, smooth, lobular, and of the consistence of adi-ose tissue. Very rarely are they somewhat pedunculated. The skin ver them is normal except for occasional increased pigmentation. The umors occur in the subcutaneous tissue, and the skin over them is ovable, unless there has been an accidental inflammatory process and econdary adhesions to the capsule of the tumor. The tumors themselves re also movable. There may be one or several tumors in a given case, ut they are not usually very numerous. Their commonest location is he neck, the back, and the buttocks. From their prominence, circum-ribed lipomata are readily injured, and they not infrequently become nflamed and ulcerate.

Diffuse lipomata occur as ill-defined fatty swellings which may in-olve large areas. They do not differ from normal accumulations of fat ccept in their irregular outline and asymmetrical distribution.

The term FATTY NECK is applied to a form of diffuse lipoma which nvolves the neck in fatty, sometimes nodular, tumors which may reach ery large size.

The growth of lipomata is slow, and after a certain time it usually ases. Occasionally circumscribed lipomata disappear as the result of lceration or inflammation. Very rarely do they become the site of alignant degeneration.

Both circumscribed and diffuse lipomata are without subjective mptoms.

Etiology and Pathology.—The cause of lipomata is unknown. hey occur most frequently in later life, but in a few cases they have een congenital. The circumscribed form is most common in women, e diffuse in men. The histology of lipomata differs very little from that of normal cutaneous adipose tissue aside from the fact that the fat cells are, as a rule, larger. A lipoma consists of a local growth of fat tissue about an arteriole and its branches, this distribution of its growth accounting for the characteristic lobulation of the tumors. The fat is, of course, supported by a stroma of connective tissue. In the circumscribed growths this is condensed at the periphery into a capsule sharply lineating the tumor from the surrounding tissue. In diffuse lipoma the capsule is incomplete or lacking.

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[1] Brodie, "Lectures on Pathology and Surgery," 1846.—Baker and Bowlby, *rans. London Med. Chir. Soc.*, 1886, vol. lxix, p. 41.

Lipomata are subject to various degenerative changes such as calcareous, myxomatous, or colloid. Occasionally there is an increase of the vascular tissues producing the so-called lipoma telangiecticum.

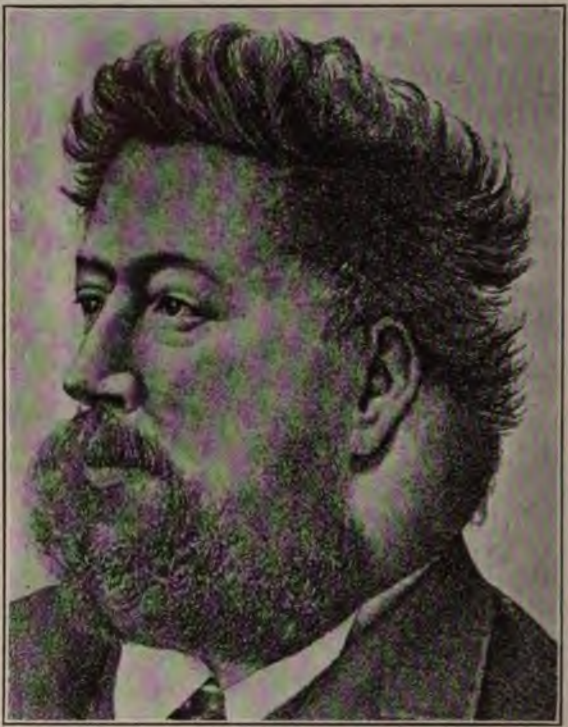

Fig. 274.—Diffuse Lipoma of Neck (Fatty Neck). (Photograph of Plate in Hutchinson's smaller Atlas.)

Diagnosis.—In both diffuse and circumscribed lipomata the fact that the tumors are of the consistence of adipose tissue, are movable, lobulated, noncompressible, slow-growing, and painless, suggests their character. Circumscribed lipomata are most likely to be mistaken for sebaceous cysts. These are rounded and not lobular.

Treatment.—Circumscribed lipomata are very readily removed by excision. In diffuse lipomata this procedure is not practicable, and they are ordinarily beyond relief. Thyroid extract is sometimes of benefit in diffuse lipoma, including fatty neck.

Adiposis Dolorosa.[1]—Under the term adiposis dolorosa, Dercum has described cases of an affection occurring in middle-aged women, racterized by the formation of large, diffuse, fatty infiltrations in the cutaneous tissue resembling diffuse lipomata, and accompanied by n and tenderness. The fatty deposits were symmetrical, and except their pain presented the clinical picture of extreme obesity. There great muscular weakness, disturbances of sensation, nerve tender-s, and other evidences of disturbed innervation. The skin, as a rule, the cases was dry and there was often purpura, as well as hæmorrhage m the mucous surfaces. The affection suggests in some of its features xœdema, but the clinical picture presents many differences and con-nt changes in the thyroid have been lacking.

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[1] Dercum, *Amer. Jour. Med. Sci.*, 1892, p. 521.

In a case of Dercum's disease, Bondet[1] has seen great improvement in general health, and marked diminution in the lipomata from the nbined use of x-rays, thyroid extract, and potassium iodide.

VII.—MYOMA[2]

SYNONYMS: *Dermatomyoma, Leiomyoma, Muscle Tumor.*

Dermatomyomata may be divided into two kinds: first the simple or erficial, second the deep or dartoic (Besnier). The first arise from arrectores pilorum, or from the muscular coats of the blood vessels, l are multiple. The second develop from the tunica dartos, or from bryonic remnants in the skin, and occur as single tumors.

Symptomatology.—Multiple myomata of the skin are very rare, s than a score of cases being recorded. They occur as pinhead to pea e, or larger, soft, elastic tumors of reddish to brownish-red color, the or disappearing upon pressure. Rarely they are of a translucent yel-rish color. The lesions develop slowly, but persistently increase in mber and size. Involution may rarely occur. They are always mul-le, and show a strong tendency to irregular grouping. Their number usually limited, but in a few cases they have been very numerous. the limited cases there is some predilection for the face, neck, and nk, but they may occur anywhere. In the cases with numerous lesions ir distribution is irregular and without symmetry, but still there is a dency to grouping. The lesions may be painless, but in about half of cases they have been painful, the pain often, however, not appearing til the lesions were well developed. The pain is often spontaneous and roxysmal and may be severe, it is excited by pressure, and at times by osure to heat and cold, especially the latter.

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[1] Bondet, *Lyon médical*, 1904, vol. xi, p. 223.

[2] Crocker, *Brit. Jour. Derm.*, 1897, pp. 1 and 47 (review of recorded cases).—rzog, *Jour. Cutan. Dis.*, 1898, p. 527.—C. J. White, *Jour. Cutan. Dis.*, 1899, p. 266. eslie Roberts, *Brit. Jour. Derm.*, 1900, p. 115.—Hardaway, *Jour. Cutan. Dis.*, 4, p. 375.

The deep-seated myoma occur as sessile or rarely pedunculated
tractile tumors of the size of a hazelnut to a walnut or
larger. They are nearly always single, and occur where the

[illegible]

[illegible]

[illegible]

[illegible]

Prognosis and Treatment.—The lesions if removed surgically do not tend to return, although in some of the cases pain has persisted. Removal is easy when the lesions are not numerous.

VIII.—OSTEOSIS CUTIS

Coleman[1] reports a case from the practice of Sherwell of osteosis of the skin of the foot. The affection was characterized by a diffuse thickening of the skin and subcutaneous tissue of about one third of the external plantar surface of the left foot, and there was a club-shaped thickening of the fourth toe. The plaque embedded in the skin was of cartilaginous hardness, and had upon its surface about six small nodes. The epidermis was somewhat thickened, but was otherwise unaltered. The lesion interfered with walking to such an extent that excision was required, and on section there was found a general infiltration of the skin with cancellous bone for which the term osteosis was suggested rather than osteoma, because there was not a distinct tumor. Salzer has reported a case of the formation of bone in the scalp. No other cases of bone formation have been recorded. My associate, F. G. Harris, has seen bone formation in a scar of a laparotomy wound which was excised on account of an apparent keloid formation. On section cancellous bone trabeculæ occurred deep in the scar which was made up of dense fibrous tissue.

IX.—PAPILLOMA

The term papilloma has been used in the past as an appellation for various papillary growths in the skin, such as warts, small nævi, condylomata. Virchow, Auspitz, Unna, and others have shown that these growths in which there is an apparent papillary hypertrophy are really due to epithelial hypertrophy, for which Auspitz's term, acanthoma, is a better designation. The term papilloma of the skin is falling into disuse, and describes no clinical entity.

Papilloma area elevatum of Beigel was, as Crocker suggests, probably a bromide eruption in infancy.

Papilloma neuroticum is a term applied to linear nævus.

X.—DERMOID CYST

Dermoid cysts in the skin are, as a rule, indistinguishable in appearance from small fibromata. They may simulate sebaceous cysts; indeed, it is probable that in many of the cases of so-called multiple sebaceous cysts the lesions are really dermoid cysts. In a case of multiple dermoid cysts reported by Pollitzer the lesions had the appearance of xanthoma. Dermoid cysts in the skin are usually single, but they may be multiple. They vary in size from a pinhead to a hazelnut, and occasionally are much larger. They can only be differentiated from the other tumors which they resemble by the structure and contents of the tumors. Their contents consist of epithelial cells and *débris*, and frequently hairs, and, like other dermoids, their walls show the papillary layer and other structures of the skin more or less definitely developed.

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[1] Coleman, *Jour. Cutan. Dis.*, 1894, vol. xii, p. 185.

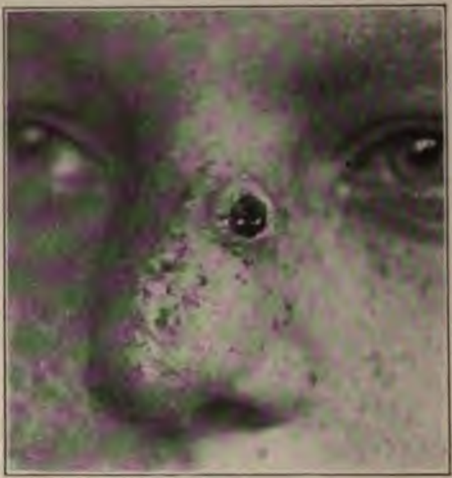

FIG. 276.—SUPPURATING DERMOID OF SKIN. From this cyst about 300 normal hairs of the size of eyelashes from light to brown color were obtained. (Author's collection.)

(*A*) *TUMORS OF THE BLOOD VESSELS*

Modifying the classification of Unna and Pollitzer, tumors of the blood vessels may be divided into:

(1) Angiectases, dilatation of existing vessels;

(2) Angiomata, proliferation of vascular tissue.

Both of these may be either congenital or acquired. The congenital forms are included under the various forms of vascular nævus. The acquired forms are telangiectases, papillary ectases usually found only in old age, and hypostatic ectases or varicose veins.

XI.—NÆVUS VASCULARIS[1]

SYNONYMS: *Nævus vasculosus, Angioma, Nævus sanguineus, Mother's Mark, Birthmark, Port Wine Mark, Feuermal, Tâche de feu.*

A vascular nævus is a congenital hyperplasia of a circumscribed area of the vascular system of the skin. This hyperplasia varies enormously in different cases (from superficial capillary dilatation in the simplest cases to large, cavernous angiomata in the most extreme cases) and the clinical pictures which are produced by nævi show all grades of intensity between these extreme limits. According to the degree of development, vascular nævi may be divided into three forms:

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[1] Anthony, *Jour. A. M. A.*, June 18, 1904.

First, Flat Nævi, in which the nævus consists of a superficial plexus of dilated capillaries; this form includes cases described under various names, like nævus angiectodes, nævus flammeus, port wine mark.

Second, Hypertrophic Nævi; these are well-defined angiomata made up of a network of large, dilated blood vessels. Under this form are included angioma simplex hyperplasticum (Virchow), angioma plexiforme (Winiwarter), angioma glomeruliforme (Unna).

Third, Angioma Cavernosum of Winiwarter, which is a cavernous nævus of steady growth, destroying the surrounding tissues by mechanical interference.

Symptomatology.—In all of these forms the symptoms are chiefly those arising from abnormal vascularity of the skin. The color varies between bright red and dark purple, according to the arterial or venous character of the blood. The lesions are compressible. In the superficial nævi the redness disappears momentarily on pressure. When the nævi are large they collapse with a sensation a good deal like that in the compression of a sponge. Over the most superficial lesions the skin is without elevation and may be quite smooth. Ordinarily it is slightly roughened, presenting a texture a good deal like that of an orange skin. Occasionally it is warty and thick.

The superficial, flat nævus occurs as an area of redness produced by a network of dilated capillaries. On close examination the formation of the lesion from a plexus of dilated capillaries can readily be made out with the naked eye. The color is usually a bright red, of different intensity in different cases according to the degree of vascularity of the area; occasionally the color is dark red or even bluish red. The border in this, as in other forms of nævi, is sharp, but there may be small, satellite nævi around the borders of the chief lesion or radiating irregularity from it, and sometimes there are minute lesions like cayenne pepper grains about the border. The shape of the lesions is quite irregular, and there is practically no limit to their size. They may be smaller than a coin, and ordinarily they do not cover an area larger than a hand, but to this there are many exceptions, and in extreme cases they spread over such large areas as the entire side of the face or head or the entire length of a limb. Their most frequent locations are the face and head, but they may occur anywhere. The commonest site is said to be the back of the head just below the occipital prominence, but the cases which come under the observation of dermatologists are most frequently on the lip, the cheek and side of the nose, and the glabella. They are usually single or composed of several nævi grouped in one small area. Occasionally there are several at different sites. In a remarkable case recorded by Pollitzer the body was covered with closely set, small, flat nævi of the average size of a dime. Except at the back of the head, they are usually distinctly unilateral, rarely spreading beyond the median embryonic fissures.

The hypertrophic nævi present the same characteristics as the flat, but of greater degree, due to the larger size and greater number of the blood vessels. In the lesser degrees of this form the surface is slightly or not at all elevated, somewhat roughened and uneven, with perhaps some evidence of lobulation. In cases of more pronounced degree the surface is irregular and nodular, and the lesions elevated. The extent of the lesions vary from a single lesion of the size of a pea or hazelnut to large lobular tumors involving the entire side of the head and neck. In all of these lesions anything that momentarily impedes the circulation, such as coughing or crying, causes temporary congestion of the lesions. In the more pronounced lesions pulsation is often apparent, particularly if they are located over bone. Hypertrophic nævi occur most frequently on the head and neck. Either flat or hypertrophic nævi may be present at birth or make their appearance a few to several weeks after birth. They usually increase more or less rapidly for several weeks, and then become of fixed size. In rare cases the hypertrophic lesions extend widely. The lesions of extreme degree sometimes ulcerate through the mechanical interference which they produce in the circulation of the overlying tissues. In this way nature may produce a cure, but the process may be accompanied by alarming hæmorrhage, and death may result from the cachexia produced by the hæmorrhage and ulceration. This result, however, is very rare. Both forms of vascular nævus are said frequently to undergo involution without ulceration, but this in my opinion is very uncommon. When evolution without ulceration occurs, there is scarring, which is slight and atrophic in the most superficial cases, and deeper and more irregular in the most pronounced lesions. Both of these forms of nævus are, as a rule, unaccompanied by subjective symptoms.

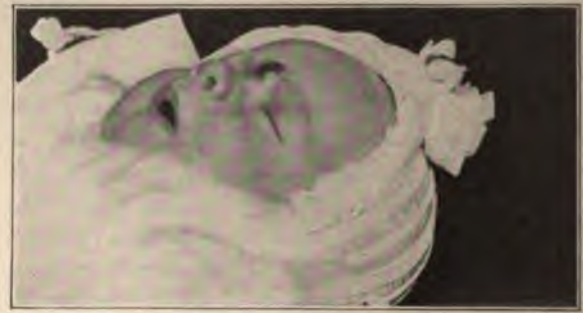


FIG. 277.—VASCULAR NÆVUS. (Author's collection.)

Cavernous nævus, or the angioma cavernosum of Winiwarter, appears as a globular or lobulated tumor of purplish color. It is more deeply seated in the tissues than the other forms, and its peculiar characteristic is its persistent but slow growth. Cavernous nævi may after a time become stationary, but they are apt constantly to progress and cause destruction of contiguous tissues, even of cartilage and bone. This effect is mechanical—analogous to that produced by aneurism—and is often accompanied by severe pain. According to Winiwarter the structure of these lesions is very closely similar to normal, cavernous tissue like that of the penis. Occasionally the cavernous nævi are erectile. Cavernous nævi may appear any time during the first year after birth, and their appearance is frequently associated with injury.

Etiology and Pathology.—According to the statistics of Depaul and of Pollitzer, nævi are present in about thirty-three per cent of newborn infants, but this far exceeds the frequency of well-defined vascular nævus. According to Gessler, vascular nævus is twice as frequent in females as in males. Such a variation between the sexes is hard to explain and is probably accidental in his statistics.

Nævi are a developmental defect, but the causes of their occurrence are altogether obscure. They appear sporadically, without any well-defined hereditary tendency. Maternal impressions are frequently invoked to explain their occurrence, but are almost surely without any influence. Virchow first called attention to the anatomical relation of nævi to the embryonic fissures of the skin, and suggested the possible influence of slight irritative conditions in producing great vascular development at the borders of these fissures where the blood vessels are very abundant. To this Unna has added the influence of pressure *in utero* as an exciting factor, and calls attention to the frequency of the occurrence of the lesions upon sites especially exposed to pressure, like the occiput, but the suggestion is purely hypothetical.

A vascular nævus consists essentially of a new growth of anastomosing, dilated capillaries which are often so dilated as virtually to form sinuses. It begins most frequently in the superficial layers of the corium, but often begins in or extends to the subcutaneous tissue. A nævus is a distinct abnormal growth. Ribbert has shown that a nævus may be injected through its underlying vessels without producing any injection of the normal blood vessels of the overlying skin and surrounding tissues. It thus has no connection with the normal blood vessels of the surrounding skin, and does not represent a dilatation of the local capillary vessels. It receives its blood supply from the underlying vessels. According as it is in more intimate connection with the arteries or veins, it may be distinguished as arterial or venous.

The cavernous nævus differs from the ordinary nævus only in having large blood spaces instead of simple dilated capillaries.

Diagnosis.—The diagnosis of vascular nævus offers no difficulty. The presence of an angioma at birth or developing quickly afterwards is characteristic. A hæmatoma could only be confused with a nævus by the most careless examination, and its character would be declared by its subsequent course.

Prognosis and Treatment.—Vascular nævi, although they occasionally disappear spontaneously, are usually permanent. They may enlarge slightly and then remain fixed, or they may increase very greatly in size. For this reason it is better to operate on them early, unless after a few weeks they show no tendency to grow.

The treatment of vascular nævi, except those of small extent, is difficult, and is followed by only partially successful cosmetic results.

Where the lesions are small and can be destroyed by excision or other destructive measures without producing great deformity, they can be entirely replaced by white scar tissue. Where they are large, the best result that treatment can produce is the more or less complete replacement of the lesions by scar tissue. Where the lesion is not small enough to make excision practicable, the first method of treatment to be tried should be compression of the lesion with the hope of exciting the spontaneous involution that sometimes occurs. This procedure is best carried out by keeping the surface covered with plain collodion. Where this is not sufficient, mechanical pressure by the application of a pad under a bandage may be tried.

Where complete excision is not possible, the only measures available are those which undertake to destroy the blood vessels without complete destruction of the surrounding tissue. Many procedures are used. For superficial lesions composed of small dilated capillaries, electrolysis can be used with considerable benefit. The needle attached to the negative pole is inserted into the corium parallel with the surface and left for a few seconds, until a blanched line is produced in its course. This procedure requires a current of 2 to 5 milliampères. The object is to produce a line of scar tissue with destruction of the capillaries along its course. The surface is thus gone over with innumerable parallel insertions, and after it has been treated in one direction and the reaction has entirely subsided, the lines may be made in the opposite direction. The treatment is excessively tedious, but with care and patience good results can be obtained in superficial thin lesions. Electrolysis is also used for hypertrophic lesions. In such cases a stronger current must be used—10 to 20 milliampères. The needle is introduced into the substance of the tumor, and firm clotting produced. The procedure is painful, and in young children requires an anæsthetic. The connections between the hypertrophic nævi and the underlying vessels are sometimes through openings large enough to allow a clot of dangerous size to enter the irculation, and I know of one case of death in an infant from treating hazelnut-size angioma of the lower lip by electrolysis. While the dan-er is very remote, it exists and renders this method of treating hyper-ophic nævi less desirable than excision. This danger does not attach the use of electrolysis in the flat lesions composed of enlarged capil-ries.

Another method somewhat similar to electrolysis consists in the in-rtion into the nævus at various points of a needle dipped in nitric cid or carbolic acid. It is not preferable to electrolysis.

In flat nævi improvement can be produced by exposures to x-rays, he exposures being cautiously carried out for several weeks with the roduction of a slight dermatitis. The result is not entire removal of he lesions, but in several cases in my experience there has been very reat improvement. I have seen the method tried also in a hyper-rophic nodular nævus, and there was some improvement, but in such case the method in my opinion offers little prospect of material enefit.

Great improvement may also be produced by freezing with liquid air or solid carbon dioxide. A small area, from one half to three fourths of an inch in diameter, is frozen by the application of liquid air or solid carbon dioxide for ten to twenty seconds. The depth of the freezing depends upon the amount of pressure exerted and requires some care to gauge it correctly. After the freezing there is violent reaction which persists for about ten days, and is followed by very slight scarring and destruction of all or a great part of the blood vessels. In the treatment f large lesions much care has to be taken to produce the same amount f scarring in each of the divisions treated at one time.

I have in the last three years treated many vascular nævi with frozen O_2. In elevated lesions where there is some excess of tissue the results re best, and practically no visible scar may be left. In flat port-wine arks great improvement may be produced. This method and freezing ith liquid air offers a means of dealing effectively with vascular nævi. t is incomparably better than the older methods, and I believe is better han treatment with radium, as suggested by Wickham. There is now bundant testimony as to the beneficial results from treatment of ascular nævi by refrigeration with solid CO_2.

XII.—TELANGIECTASIS

Etymology: τέλος, end; ἀγγεῖον, vessel; ἔκτασις, dilatation.

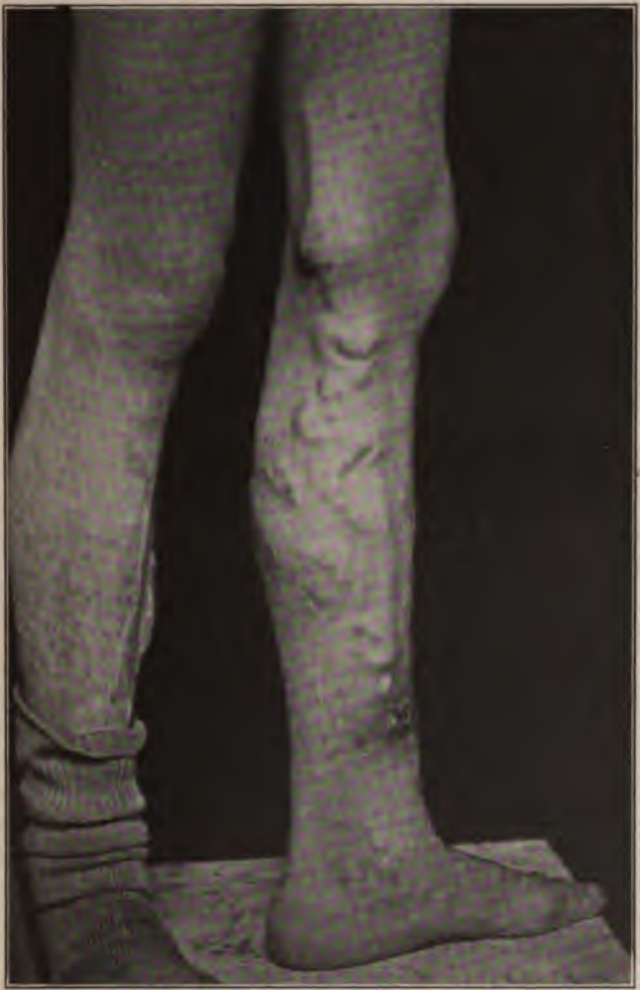
Telangiectases are acquired dilated capillaries in the skin. They ccur as tortuous or wavy red lines, which may be made to disappear on pressure and are manifestly dilated blood vessels. There may be only a few of them, as is frequently seen in the naso-labial furrows, or they may be so numerous as to produce a retiform mottling of a large surface. Anastomoses among the blood vessels are common. Occasionally telangiectases occur in the form of a central red dot with radiating dilated capillaries. This constitutes the so-called *spider nævus*, or *nævus araneus*. Such lesions may be congenital and thus true nævi, or they may be true telangiectases.

Telangiectases occur secondarily in various diseases, among which are morphea, and tubercular, syphilitic, and malignant infiltrations of the skin. In all of these conditions they may be regarded as a manifestation of a functional hypertrophy for the purpose of meeting the demand of a lesion for an increased blood supply. They also occur as a primary symptom in rosacea, xeroderma pigmentosum, and the flushed type of senile skin.

Primary telangiectases are frequent on the flush areas of the face (the cheeks and nose) in certain individuals with florid faces who also have a tendency to senile keratoses. An out-of-door life and old age are both factors in the development of this primary type of telangiectasia, but the most important factor is individual peculiarity which is often hereditary. Primary telangiectases, unassociated with other changes in the skin, are frequent as a result of peripheral circulatory disturbances. They are thus common on the outer surfaces of the thighs, on the abdomen, especially at the lower borders of the ribs, and over the shoulders. Those on the nose are sometimes produced by intranasal obstructions to the circulation, but are most frequently the result of gastrointestinal disturbances which cause habitual flushing of the face. A most extreme form of telangiectasia results at times from x-ray effects upon the skin. This is usually seen after acute dermatitis, but may result from long-continued slight reaction in the skin. Its occurrence is apparently explained by the fact that in x-ray dermatitis there is an obliterating endarteritis which destroys many of the capillaries and results in compensatory hypertrophy in many of those remaining.

Histologically telangiectases are simply dilated capillaries.

Treatment.—Telangiectases on the face, especially about the nose, frequently result from the same factors that produce rosacea. These are in most cases disturbances of digestion. Sometimes those on the nose are due to obstructions to the venous flow by lesions in the nose. The further progress of these cases may be affected by the general treatment of the conditions which produce them, but the telangiectases already formed are permanent, and their removal requires local destruction. They may be very readily destroyed by electrolysis. The needle attached to the negative pole is threaded for one eighth to one fourth of an inch into dilated vessel and a current of from two to four milliampères is ed until the vessel is blanched. In this way a few vessels can be royed at each sitting and a satisfactory result obtained by repeated



IG. 278.—Varicose Veins and Small Ulcer of Leg. (Author's collection.)

tments. The same result may be obtained by slitting up the telan- tases for a fourth of an inch and cauterizing them with carbolic acid.

Papillary Ectases

The papillary dilatations, or ectases, of the old are red spots from ixteenth to half an inch in diameter composed of dilated blood vessels. They are found on the trunk and upper parts of the thighs, and frequently occur before old age. They are usually classed as ectases, but are really new growths—angiomata (Ribbert).

GENERALIZED TELANGIECTASIA, HEREDITARY HÆMORRHAGIC TELANGIECTASIA, TELANGIECTASIA CIRCUMSCRIPTA UNIVERSALIS

Various writers have described cases of generalized or multiple telangiectasia, either hereditary or acquired. The lesions occur especially on the face, and often on the buccal and nasal mucous membranes. The nasal ectases frequently manifest their presence by epistaxis, particularly during childhood. An hereditary tendency is often found in the condition, which in many cases is a congenital defect. In other cases the condition is acquired and is the result of the same group of factors that are recognized as causing the development of the usual telangiectases. The cases are, there seems every reason to believe, exaggerated examples of ordinary telangiectasis, some of whose lesions are found in every person.

XIII.—ANGIOMA SERPIGINOSUM[1]

SYNONYMS: *Infective angioma, Nævus lupus* (Hutchinson), *Sarcome angioplastique réticulé* (Darier).

This is a rare affection resembling clinically very superficial nævi which, however, persistently spread at the periphery.

The lesions consist of vascular macules like "cayenne pepper grains," arranged in roundish patches. The patches tend to spread peripherally with central clearing, thus forming incomplete rings composed of red, minute points. The affection appears in early life or infancy, and is slowly progressive. The condition has appeared upon various parts—the arm, leg, foot, chest, face—without the influence of any ascertainable determining factors to account for its distribution. There have been no subjective symptoms except slight itching in White's case. The condition is excessively rare. Four of the seven cases have been recorded by Hutchinson, who first described the condition. Five of the cases began before the third year and occurred in females. Anatomically the process is a proliferation of the blood vessels which is suggestive of angiosarcoma. The treatment of the lesions by cauterization, excision, and other destructive measures has failed to check the course of the disease.

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[1] Hutchinson, *Arch. of Surg.*, vol. iii, p. 166.—J. C. White, *Jour. Cutan. Dis.*, 1894, p. 505.—Crocker, *Brit. Jour. Derm.*, 1894, p. 367.—Francis, "International Atlas," Plate XXXIV.—Schamberg, *Brit. Jour. Derm.*, vol. xiii, No. 147. (A unique case presenting clinical resemblances to angioma serpiginosum.)

(*B*) *TUMORS OF THE LYMPHATICS*

Theoretically tumors of the lymphatic vascular system may be divided into lymphangiectases (dilatations of normal lymphatics) and lymphangiomata (new growths of the lymphatic vascular tissue); the first, analogous to telangiectases and other ectases of the blood vessels, the second, to hæmangiomata. As a matter of fact, however, dilatations of the lymphatics are always associated with proliferation of the lymphatic tissue, so that a complete distinction does not actually exist. A pathological distinction, however, is readily made: lymphangiomata are practically all the result of embryonic defects. They are either congenital or they are, in all probability, the result of misplaced embryonic remnants which, either with or without some local exciting cause, subsequently proliferate. According to their location in the tissue, lymphatic tumors may be divided into superficial and deep. The superficial alone occur in the skin, and concern us here.

XIV.—LYMPHANGIECTASIS

Lymphangiectasis is a dilatation of the normal lymphatics of a part. It is always associated with the formation, to a greater or less extent, of new lymphatic vessels, so that it is in reality in a sense an acquired lymphangioma. It is always the result of some condition causing obstruction of the lymphatic vessels.

Symptomatology.—The usual form of lymphangiectasis is elephantiasis (*q. v.*). In this the lymphatic obstruction is accompanied by great overgrowth of connective tissue. As already seen, elephantiasis usually involves an entire part, as a limb for example. Occasionally it is sharply localized to a small amount of tissue, as one vulvar labium, or the scrotum. It may involve only the lips, or one lip, producing the condition known as *macrocheilia*, or it may involve the tongue alone, *macroglossia*. These conditions are distinctly forms of elephantiasis, and are only mentioned here as examples of lymphangiectasis. Ordinarily in elephantiasis the connective-tissue proliferation overshadows the lymphatic dilatations in the production of macroscopic features. In rare cases, however, the lymphangiectases are a more prominent feature, and produce cystic nodules upon the surface from the size of a pea to an apple. Such a tumor, occurring on the lower part of the abdomen and front of the upper half of the thigh, is described and illustrated by Stelwagon, and L. E. Schmidt demonstrated before the Chicago Dermatological Society a case almost identical in appearance with Stelwagon's case, involving the same area, and in my opinion of the same character, which was associated with an extensive rapidly growing tumor, probably a sarcoma, in the pelvis. These cases may be regarded as essentially of the same character as elephantiasis, but with the lymphangiectases a more pronounced feature than usual. Similar smaller cystic nodules are at times seen in macroglossia and macrocheilia.

Lymphangiectases are also at times represented by small compressible tumors which may have upon their surface transparent vesicles similar to those of lymphangioma circumscriptum. Upon puncture of the vesicles, or of the tumors, there is a free flow of lymph. The lesions may be of normal color or reddened, and usually there is some thickening of the tissues, but this may be absent. The character of the lesions can only be definitely made out by the free flow of lymph from them when they are punctured, or from their anatomical structure. Epstein[1] has recorded an interesting case of acquired lymphangioma which began in a woman about twenty-five. It consisted of glistening, pinkish, firm, papulo-vesicles from the size of a pinhead to a pea, some of them smooth and some of them warty, which developed in an abundant crop on the genitals and the lower part of the abdomen, and also on the right but-

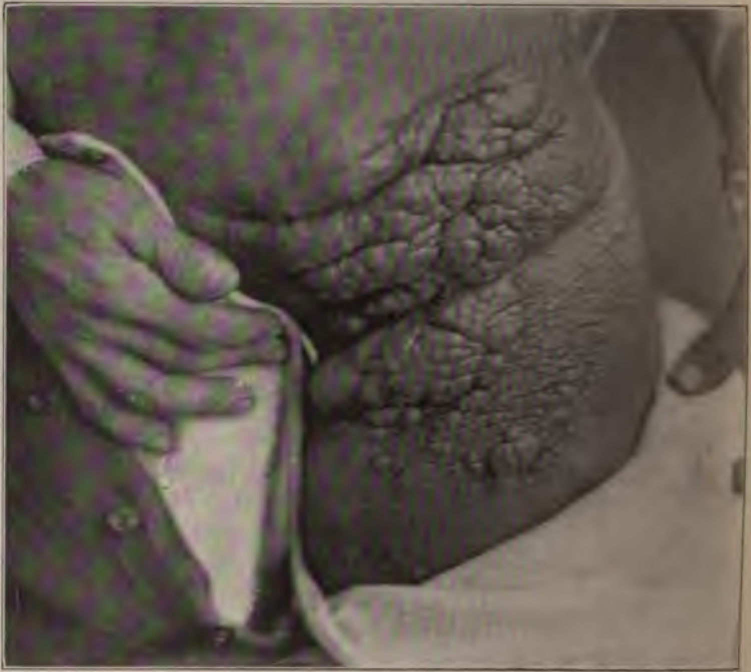

Fig. 279.—Elephantiasis from Obstruction Produced by a Pelvic Tumor. (L. E. Schmidt's photograph.)

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[1] Epstein, *Jour. Cutan. Dis.*, 1892, p. 213.

tock. They were associated with dilated blood vessels, and with a few lymphatic varices which radiated from the affected area. On puncture of the vesicles there was a discharge of pure lymph which continued to flow for several hours. The condition had existed for fourteen years at the time when the patient came under observation. There was some hypertrophy of the labia minora. It was accompanied by itching.

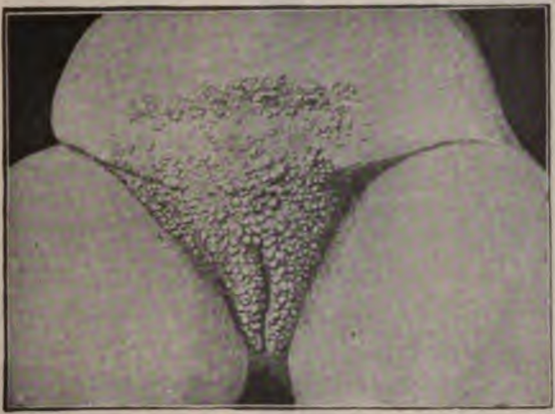

FIG. 280.—ACQUIRED LYMPHANGIOMA. (Epstein's case.)

The patient had previously had syphilis, and Epstein regarded the lymphangioma as due to syphilitic lymphangitis which had produced obstruction in the lymphatic vessels. Under specific treatment the condition had improved very much at the time of his report. The case is highly interesting as illustrating the development of lymphangioma features in lymphangiectasis without the development of the features produced by connective-tissue increase.

In a very few cases reported by Elliot,[1] Besnier, and others, simple lymphangioma, or lymphangiectasis, has developed in the skin in the form of a circumscribed patch of papulo-vesicles similar in appearance to lymphangioma circumscriptum. These cases have developed around scar tissue or after injury, and are apparently the result of mechanical obstruction of the lymphatics.

Treatment.—In all forms of lymphangiectasis treatment is usually unsuccessful because of the impossibility of relieving the obstruction producing the condition. Circumscribed lesions may be amenable to the same treatment applicable to lymphangioma circumscriptum.

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[1] Elliot, *Jour. Cutan. Dis.*, 1894, p. 137.

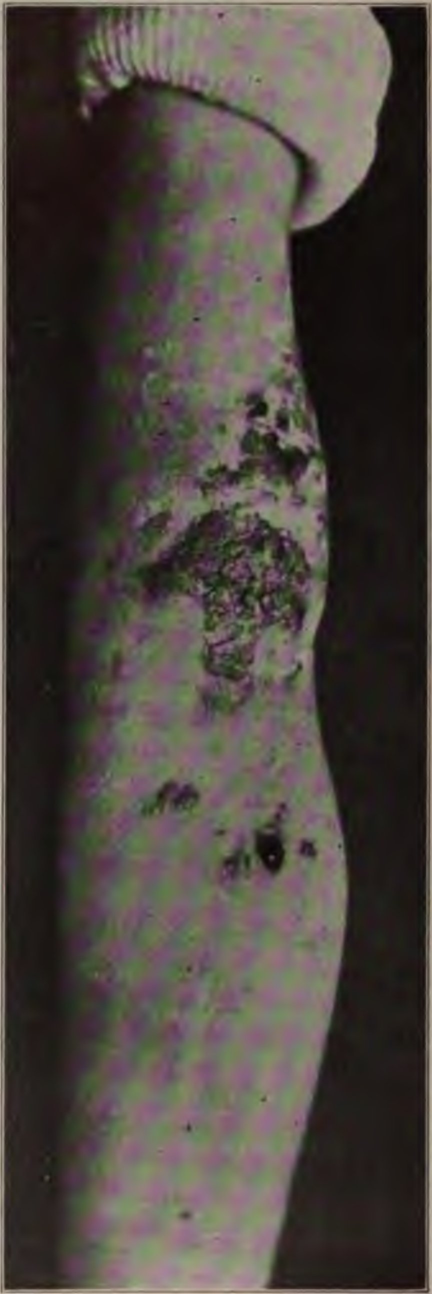

FIG. 281.—LYMPHANGIOMA CIRCUMSCRIPTUM. (C. J. White's collection.)

XV.—LYMPHANGIOMA CIRCUMSCRIPTUM[1]

SYNONYMS: *Lymphangioma sup ficium simplex* (Unna), *Ly phangioma capillare varicos* (Török), *Lupus lymphati* (Hutchinson), *Lymphang todes* (Tilbury Fox), *Lymph gioma cavernosum* (Besni *Angiome cystique* (de Smet a Bock), *Lymphangiome circ scrit vesiculeux* (Brocq and B nard).

Lymphangioma circumscr tum is a proliferation of ly phatic vascular tissue in circu scribed areas of skin analogo in all probability, to vascul nævus.

The disease was first d scribed by Tilbury and Colco Fox, under the title lymphang ectodes, and most of the cas have been reported in Englan

Symptomatology.—T condition usually occurs as permanent patch of groupe yellowish or reddish, tough pa ulo-vesicles. The vesicles a from one to two or three mil meters or more in diameter, a they are usually associated wi telangiectases, so that while th may be yellowish and transpa ent, they are often pearly reddish or pinkish in colo

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[1] Tilbury and Colcott Fox, *Lo don Pathol. Soc. Trans.*, vol. xxx, 187 —Malcolm Morris, "International A las," 1889, Plate I.—Elliot, *New Yo Med. Record*, vol. xxxix, 1891.—J. White, *Jour. Cutan. Dis.*, 1894.- Gilchrist, *Johns Hopkins Hosp. Bul* 1896, p. 138.

They have the appearance of simple vesicles, but they have a thick covering and do not rupture readily, but if ruptured there is a free flow of clear alkaline lymph. The vesicles occur in close groups, of irregular shape and of the size of a small coin. The whole lesion is made up of an aggregation of these groups, the individual groups being separated by areas of healthy skin, but all together forming a patch of the size of three or four fingers. This typical picture may present variations. In White's case the vesicles were larger, and covered by tough crusts composed apparently of coagulated lymph. In his case the vesicles were closely grouped into a large patch showing a wartlike, roughened surface. The condition is usually limited to a single patch, and this has in most cases occurred on the neck or the upper part of the trunk, but it has also been seen on the vulva, the buttocks, the arms, and the legs. The lesions are persistent, and the patches, as a rule, after reaching a fixed size remain stationary. Sometimes the individual papules disappear, but in most cases there is slight, gradual peripheral extension of the lesions. Occasionally the patches may very slowly change their location on the surface, the individual lesions slowly disappearing at one border while new ones appear at the other. Some of the cases of lymphangioma circumscriptum have had recurrent erysipelaslike attacks which were probably simply the result of infection. There are no subjective symptoms.

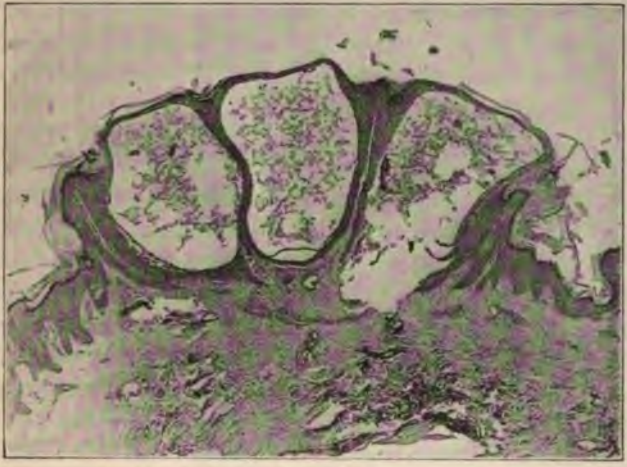

FIG. 282.—LYMPHANGIOMA CIRCUMSCRIPTUM.
(C. J. White's collection.)

Etiology and Pathology.—Lymphangioma circumscriptum begins in infancy or early childhood, and in addition to the frequent association with dilated capillaries has occasionally occurred with vascular nævi.

The lesions are true lymphangiomata of the skin—i. e., they represent new growths of a circumscribed area of the lymphatic vascular tissue, independent of the surrounding normal lymphatics (Ribbert). They are thus analogous to vascular nævi.

Histologically there is a proliferation and dilatation of lymphatic vessels, the walls of which are more or less thickened and show cystic dilatations. The lesions occur most frequently in the papillary layer, but they may involve the body of the corium, or even the subcutaneous tissue. They are often associated with telangiectases or with true hæmangiomata. The overlying epidermis may be normal or hypertrophied.

Diagnosis.—The beginning of the affection at birth or in early childhood, its occurrence in a single patch of thick-walled, grouped vesicles from which there is a free discharge of lymph upon puncture, and the association with telangiectases, distinguish the condition from most other affections. Histological examination, however, is required for positive diagnosis.

Treatment.—The patches show a marked tendency to recur, and treatment to be successful must be radical excision wide of the border of the patch. Short of this, the patches may be treated by electrolysis or such other destructive measures as are useful in vascular nævus.

XVI.—LYMPHANGIOMA TUBEROSUM MULTIPLEX[1]

Under the name lymphangioma tuberosum multiplex, Kaposi described a condition characterized by an abundant eruption of grouped papules and nodules from the size of a pinhead to that of a pea or bean, of pinkish or reddish color, and occurring upon the front and back of the trunk, being especially abundant below the clavicle. There has been much discussion as to the character of this condition. Dermatologists are pretty generally agreed that it is identical with multiple benign cystic epithelioma. Anatomically, according to Crocker, the lesions of lymphangioma tuberosum multiplex are cysts lined with flat, nucleated epithelium from which there extend ductlike cylinders of epithelial cells about the size of a sweat duct. On the other hand, Ribbert, following Kromayer and Jarisch, regards Kaposi's lymphangioma as endothelioma, composed of strings of endothelial cells. Clinically the condition differs from multiple benign cystic epithelioma chiefly in the fact that the lesions occur on the trunk rather than the face.

Van Harlingen, Pospelow, and Leslie Roberts have recorded cases of lymphangioma tuberosum multiplex which were apparently cases of fibroma multiplex.

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[1] Pospelow, *Archiv*, 1879, p. 521.—Van Harlingen, *Trans. Amer. Derm. Ass'n.*, 1881, p. 28.—Leslie Roberts, *Brit. Jour. Derm.*, 1896, p. 309.—Kaposi, "Lymphangiomata tuberosum multiplex," p. 530.

(C) *BENIGN EPITHELIAL TUMORS*

XVII.—MULTIPLE BENIGN CYSTIC EPITHELIOMA[1]

SYNONYMS: *Syringo-cystadénome* (Török), *Epithelioma adenoides cysticum* (Brooke), *Adenoma of the Sweat Glands* (Perry), *Trichœpithelioma papillosum multiplex* (Jarisch), *Hydradénomes éruptifs* (Jacquet and Darier), *Cellulome épithéliel éruptif kystique* (Quinquad), *Cystadénomes épithélieux bénins* (Besnier), *Nœvi epitheliaux kystiques* (Besnier), *Multiple benign cystic epithelioma* (Fordyce), *Hemangio-endothelioma tuberosum multiplex* (Jarisch), *Acanthoma adenoides cysticum* (Unna), *Spiradenoma, Spiroma, Adenoma sudoriparum.*

Under the various names given above, cases have been described ich consist of multiple, small, epithelial tumors of benign character urring chiefly about the face. It is evident from the names that growths develop in erent cases from dif- ent parts of the rete; some cases from the r follicles, in others n the sweat glands, others from the rete lf; the cases, how- r, form one clinical ty of fairly typical racter.

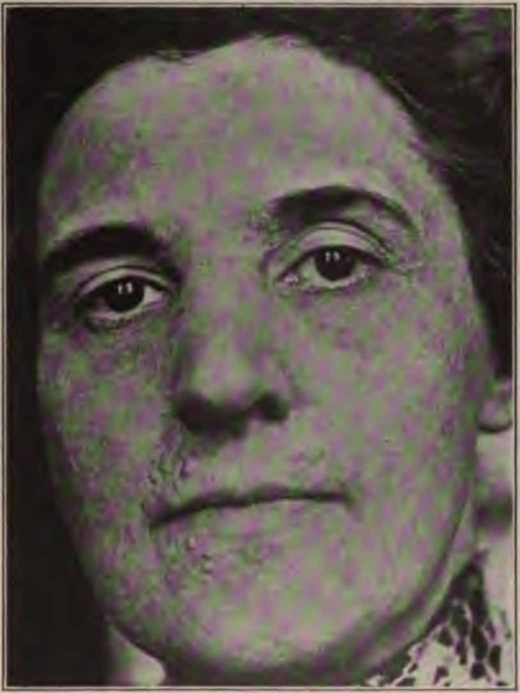

FIG. 283.—MULTIPLE BENIGN CYSTIC EPITHELIOMA (DAUGHTER). (Author's collection.)

Symptomatology. he lesions of multi- benign cystic epithe- a are papules or tu- les from the size of nhead to a pea, occa- ally larger. Their face is smooth and tening and they have translucent appear- e, so that they may k like vesicles, al- ugh they are firm and solid. They may be of a yellowish tinge, but frequently they are associated with telangiectases and are then of pinkish or pearly color. On puncture of the lesions there is simply a discharge of blood or serum, although in Dyer's case some of the lesions contained a gelatinous material. The tumors occur abundantly, and in most of the cases have a distinctly symmetrical distribution. Their usual sites are the face, especially about the lips, the naso-labial furrows, the forehead, and the ears. They also occur, though less abundantly, upon the neck and shoulders and the upper part of the arms. The lesions remain discrete, or when very abundant may coalesce into small, irregular, nodular tumors. The lesions begin as minute papules of normal color, or as black dots (Brooke), and their increase in size and number is very slow. After reaching the size of a pea the lesions usually remain stationary, but the disease as a whole becomes more abundant from the growth of new lesions. The condition shows no tendency to spontaneous disappearance. As a rule no degenerative changes occur, but in a few cases

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[1] Jacquet and Darier, *Annales*, 1887, p. 317.—Török, *Monatshefte*, vol. viii, 1889, 16.—Perry, "Adenomata of the Sweat Glands," "International Atlas," iii, 1890, e IX.—Brooke, *Brit. Jour. Derm.*, 1892, p. 269.—Fordyce, *Jour. Cutan. Dis.*, , p. 459.—J. C. White, *Jour. Cutan. Dis.*, 1894, p. 477.—Dyer, *New Orleans . and Surg. Jour.*, vol. i, 1897–98, p. 530.—Buxton, *Jour. Cutan. and Gen. Urin.* , April, 1901.—Hartzell, *Amer. Jour. Med. Sci.*, September, 1902.—*Brit. Jour. n.*, 1904, p. 361.

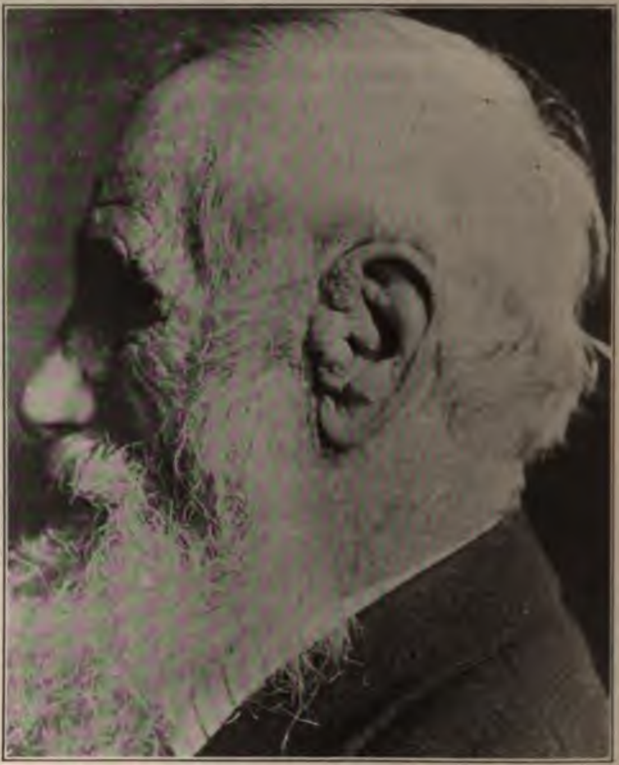

FIG. 284.—MULTIPLE BENIGN CYSTIC EPITHELIOMA (FATHER).
(Author's collection.)

l by White and others some of the tumors have degenerated into epitheliomata. Considering, however, the character of the tu- is rather surprising that this has not occurred more frequently; nple, my elder patient was a senile man, well beyond seventy, in egardless of his condition, an epithelioma would not have been a . There are no subjective symptoms accompanying the condition.

logy and Pathology.—The condition is extremely rare. Le- ay be present in childhood, but the disease usually makes its nce at puberty or during adolescence. There has been a marked ry tendency in most of the cases; groups of cases involving and children have been reported repeatedly, and in Fordyce's re was a history of the occurrence of the disease in three gen- . Multiple benign cystic epitheliomata probably represent hy- ies of misplaced ic epithelial ele- Microscopically mors are com- f communicating nching cords and of epithelial cells. centers of many cords and masses are cystlike for- of colloid or matter which is y the secretory of the cells. owths thus cor- to so-called cyl- ta, which are wing benign epi- tumors, seldom metastases, but recurrence after . Ribbert classi- ltiple benign cys- ithelioma under mata.

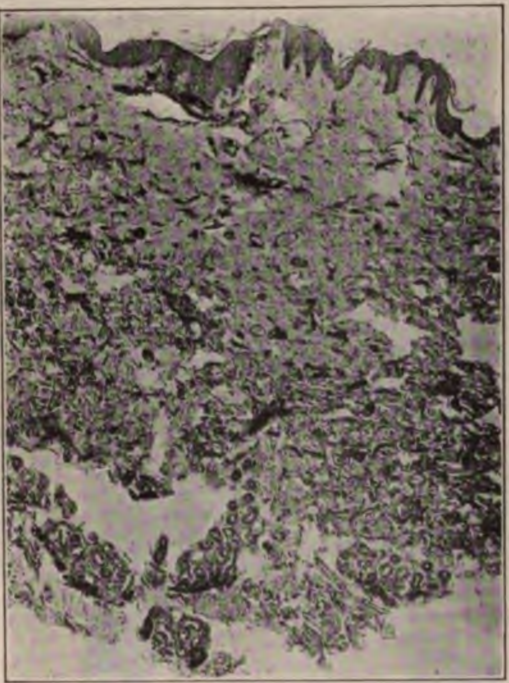

FIG. 285.—SYRINGO-CYSTOMA. (C. J. White's collection.)

gnosis.—The condition may be confused with molluscum con- n, hydrocystoma, colloid degeneration of the skin, and adenoma m. The clinical features are usually sufficient to suggest the is, but the diagnosis cannot be regarded as conclusive without a opical examination.

Prognosis and Treatment.—Individual lesions may be destroyed, preferably by electrolysis, but the lesions are so numerous that complete destruction in this way is impracticable. In my case in the daughter, the greater number of the lesions disappeared under x-ray exposures, so that the disfigurement was almost entirely removed. This result was obtained by cautious exposures scattered over a period of three months without producing any apparent reaction in the skin.

XVIII.—ADENOMA SEBACEUM[1]

SYNONYMS: *Adenoma sébacés* (Balzer and Ménétrier), *Végétations vasculaires* (Rayer), *Nævi vasculaires et papillaires* (Vidal), *Nævi symétriques de la face* (Hallipeau-Leredde).

Adenoma sebaceum is a disease characterized by the development on the face of tumors of the sebaceous glands.

Symptomatology.—The lesions of the disease are pinhead to small-pea-sized, firm, translucent tumors embedded in the skin. They may be of normal skin color, but they are nearly invariably associated with telangiectases, which color them with some shade of red. Occasionally the telangiectases are more prominent than the tumors, which may show only as minute specks in the centers of the lesions. The eruption occurs characteristically on the flush area of the face, and is usually most abundant at the sides of the nose where the tumors often become confluent. Discrete lesions are also found on the forehead, cheeks, and chin. The distribution is usually markedly symmetrical, but like other developmental defects, in some of the cases it has been sharply limited to one side. The

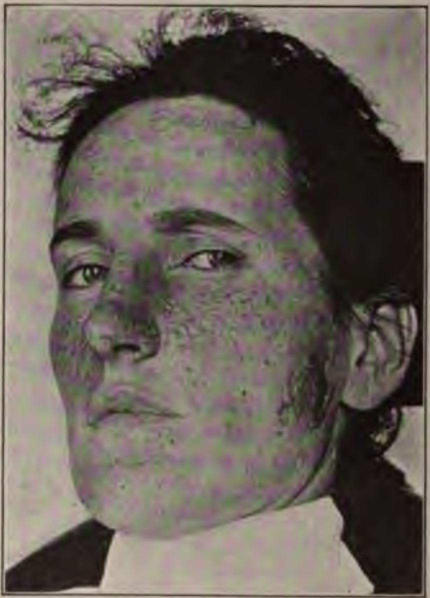

FIG. 286.—ADENOMA SEBACEUM. (Heidingsfeld's collection.)

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[1] Balzer and Ménétrier, *Arch. de Physiog.*, 1885, vol. vi, p. 564.—Pollitzer, *Jour. Cutan. Dis.*, 1893.—G. H. Fox, *Trans. Amer. Derm. Assn.* for 1898.—Gottheil, *Jour. Amer. Med. Assn.*, July 20, 1901, p. 176.—Poor, *Monatshefte*, April 1, 1905, p. 378 (review and bibliography).—Taylor and Barendt, *Brit. Jour. Derm.*, 1893, p. 360.—Poor, *Monats.*, 1905, vol. xl, No. 7 (histology and bibliography).

condition may be present at birth, or may gradually develop later. Usually its increase is most active about puberty. The individual lesions are not likely to increase above the size of a split pea, and involution of some of the lesions at times occurs, but the condition as a whole is permanent and progressive. The cases usually occur in patients with coarse skins with large sebaceous follicles, and the condition is often associated with developmental defects, such as pigmented spots, nævi, and simple fibromata.

Etiology and Pathology.—It is rare in America, but is apparently not uncommon in the pauper institutions of England. The disease is a developmental defect of the skin, and is usually congenital or begins soon after birth. In a very few cases it has developed in adult life. Most of the cases occur among the poor, and many of them are found in patients showing defects of mental development, so that the condition is most frequent in asylums for imbeciles and epileptics. The cases may, like multiple benign cystic epithelioma, occur in family groups. Stocford Taylor has reported three cases in one family.

Adenoma sebaceum is probably an error of development characterized by an adenomatous hyperplasia of the sebaceous glands, but in some cases, as in the one studied by Crocker, there is also an increase in all of the appendages of the skin. Microscopically the principal change is a marked increase in size and number of the sebaceous glands, some of which may have undergone cystic degeneration. There is often more or less vascular dilatation with increase of the fibrous tissue of the corium. The epidermis may be thickened as in Pringle's case, but more frequently is normal.

Diagnosis.—The condition is most likely to be confused with multiple benign cystic epithelioma. Confusion is also possible with the diseases enumerated as presenting some resemblances to multiple benign cystic epithelioma.

Treatment.—The only effective treatment is destruction of the lesions. They may be destroyed by electrolysis or excision. It is highly probable that they could be made to disappear by exposure to x-rays.

C. MALIGNANT NEOPLASMATA

I.—CARCINOMA CUTIS

Carcinoma of the skin, as distinguished from epithelioma, is carcinoma occurring as a result of carcinoma of the deeper tissues. It is nearly always secondary to carcinoma of the breast, rarely it is secondary to carcinoma of the visceral tissues. When it consists chiefly of nodules primarily involving the skin it is called carcinoma lenticulare (lenslike); when it consists of subcutaneous nodules not primarily involving the corium it is called carcinoma tuberosum. The distinctions are arbitrary and indicate no difference in pathological character.

Varieties. **Carcinoma Lenticulare.**—Lenticular carcinoma begins in small, hard nodules in the corium from the size of a pinhead to a pea or larger. They are firm and waxy-looking, and usually show numerous telangiectases over and around them. They involve the skin, which is tense and glistening, and they are of yellowish-pink to reddish color. The lesions may be movable on the underlying structure, but frequently they are attached to the underlying bone. The condition usually begins by the development of a few nodules in or near the scar after amputation of the breast. Sometimes they begin around the borders of carcinoma of the breast which, without operation, has involved the skin. In either case, after once developing their future course is the same. The carcinoma cells are carried along through the lymphatics of the corium,

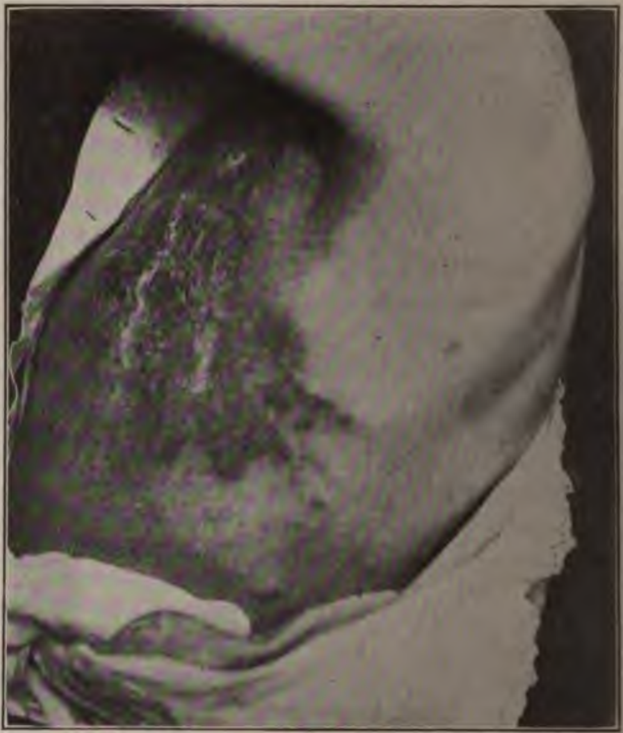

FIG. 287.—CARCINOMA CUTIS. BEGINNING CUIRASSE. (Author's collection.)

nd as they lodge new nodules appear. The lesions in the skin thus apidly increase in number, so that more or less of the surface of the hest becomes dotted with the small nodules between which the skin ay be normal or pink and more or less indurated. Adjacent nodules ay coalesce and ultimately ulceration usually occurs. The disease, owever, may spread very extensively with very little ulceration. In xtreme cases the entire chest, front, and back may be involved in a rd, boardlike carcinoma which seriously interferes with the move-ents of the chest (*cancer en cuirasse* of Velpeau). I have seen the mplete encircling of the chest from primary carcinoma of both breasts ithout any ulceration taking place. Contrary to the usual statement, ore or less cuirasse formation is not uncommon. In many instances e patients are carried off in recurrent carcinoma in the skin by deep-ated metastases before the skin involvement becomes extensive; fre-ently, however, the disease becomes widespread on the chest wall fore the fatal issue occurs.

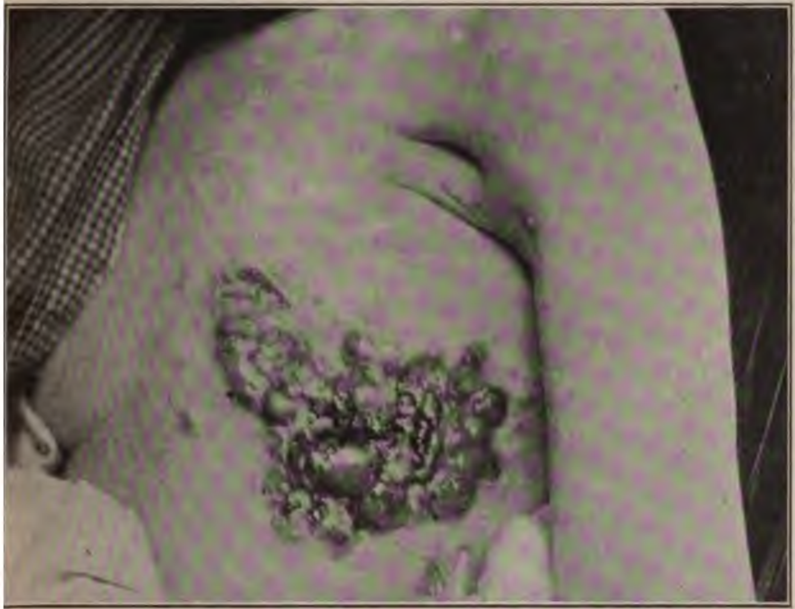

FIG. 288.—CARCINOMA CUTIS. (Author's collection.)

Secondary carcinoma of the skin is often accompanied by immediate volvement of the axillary lymphatic nodes, and of the intercostal and e supraclavicular nodes. The obstruction of the axillary lymphatics ten produces œdema of the arm, which may become extreme as the sease progresses.

Carcinoma Tuberosum.—In this form the disease occurs in nodules in the subcutaneous tissue which do not primarily involve the skin. The lesions are firm, rounded nodules from the size of a pea to a walnut or larger, usually movable at first but later becoming attached to the bony parts. The skin at first over the nodules is normal in color; later, if it becomes involved in the tumors, it becomes glistening pink to purplish in color, and presents the appearance of the lenticular lesions. The subsequent course of the lesions is then the same as that of cutaneous nodules. This form of carcinoma of the skin is sometimes primary. It is also the form which carcinoma assumes in the skin in general carcinomatosis from visceral carcinoma.

Recurrent carcinoma in the skin is accompanied by little pain so long as it involves the skin and subcutaneous tissue alone. When the bones, the tissues of the chest wall or of the shoulder or axilla become densely infiltrated, the pain is severe.

Pathology.—In secondary carcinoma of the skin, the secondary growths begin in the corium or subcutaneous tissue, and are of the same nature as the primary lesion. The nodules consist of masses of cells which extend in various directions along the lymphatic spaces.

Diagnosis.—The diagnosis of the lesions ordinarily offers little difficulty, because of their development subsequent to carcinoma of the breast, usually after operation. The development of any firm nodule in the area involved in a previous carcinoma of the breast should be looked upon with suspicion. Usually when the lesions are recurrences positive diagnosis can be made without microscopical examination, but a microscopic examination will always clear up the doubts.

Prognosis and Treatment.—Recurrent carcinoma in the skin is always of grave import. If the involvement of the skin is extensive, especially if there is palpable involvement of the axillary or supraclavicular glands, the chances of radical relief are very small. Even in these cases, however, much temporary benefit can be obtained by treatment with x-rays, and often this benefit continues for many months or even several years. In the less extensive cases x-rays may at times produce symptomatic cures. I have some symptomatic cures of this sort which have lasted now more than five years. X-rays are the only means which offer much prospect of benefit in these cases; occasionally benefit is obtained by excision of a few nodules, but unless this is followed by x-ray exposures the benefit is likely to be of very short duration.

II.—EPITHELIOMA[1]

SYNONYM: *Epidermal Cancer.*

Epithelioma is carcinoma beginning in the epithelial structures of the skin.

The term epithelioma is used in pathology with varying significance. Strictly, it is applicable to any epithelial tumor whether benign or malignant. General usage in dermatology, however, confines the term to malignant epithelioma of the skin, and it is in that sense that the term is used in this book.

Epitheliomata are among the frequent and most important lesions of the skin. They are usually divided into *superficial discoid, papillary,* and *deep,* but the divisions are based upon clinical differences and indicate no essential distinction in the character of the different varieties.

Symptomatology.—All epitheliomata have certain pathological characteristics in common.

First. They represent a purposeless proliferation of epiblastic cells which tend to extend beyond their normal limits and to invade surrounding tissues in which they are not normally found.

Second. This invasion into the connective tissue takes place through the lymphatic spaces, and is accompanied by inflammatory changes. The cause of the inflammatory process is not settled. Possibly it is due to elaboration by the pathological cells of a poison which acts as an irritant, and at the same time lowers the resistance of the involved tissues. The simpler and more probable explanation is that the inflammation is, in large part at least, the result of the irritation produced by the masses of carcinoma cells acting as foreign bodies.

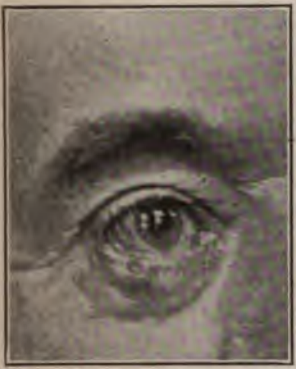

FIG. 289.—MINUTE EPITHELIOMA OF CILIARY BORDER. (Author's collection.)

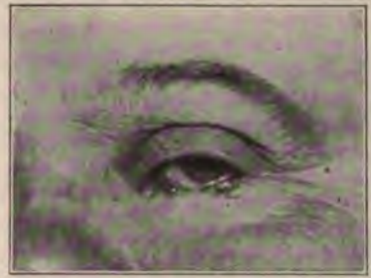

FIG. 290.—SUPERFICIAL EPITHELIOMA WHICH HAS DESTROYED LOWER LID. (Author's collection.)

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[1] Fordyce, *Jour. Cutan. Dis.*, April, 1902.

Third. The carcinoma cells occur in masses having an alveolar arrangement.

Fourth. The carcinoma masses tend to undergo necrosis and form ulcers. This is due to the fact that the blood vessels do not extend into carcinoma tissue proper, and thus by their overgrowth they cut off their own supply of nutrition.

Fifth. There is always the danger of the transference of carcinoma cells through the lymphatic vessels and the lodgment of these cells in contiguous lymphatic nodes or elsewhere with the production of metastatic growths.

All forms of epithelioma present certain clinical features in common. The lesions may begin as small, superficial plates of induration in the skin, or as small, superficial tubercles, or as hard, deep, subcutaneous tubercles which only involve the surface of the skin after considerable growth. However they begin, as soon as the skin is involved, there is evidence in the lesions of abnormal epithelial proliferation. If the lesions are small, intact tubercles or plaques, they show as firm, waxy, pseudo-translucent masses over which the skin is stretched and shiny and of pinkish or yellowish color. These lesions, in fact, present the same characteristics as the nodules of carcinoma of the skin which are secondary to carcinoma of the breast. The firmness and the yellowish or pinkish waxy appearance of carcinoma tissue are extremely characteristic features. The firmness is said to be sometimes lacking, but this is uncommon. While the tissue is firm to the feel, it is very friable and breaks down readily when curetted or exposed to any mechanical force.

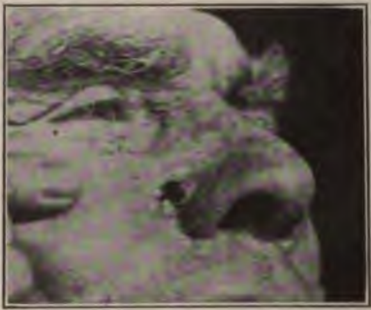


Fig. 291.—Small Craterform Epithelioma. (Author's collection.)

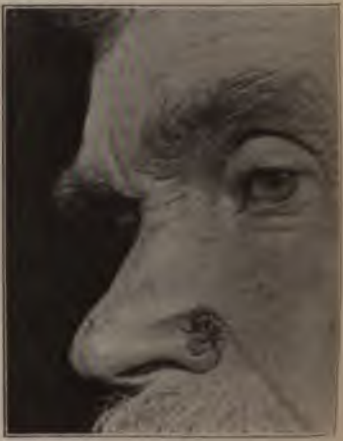


Fig. 292.—Superficial Discoid Epithelioma. (Author's collection.)

When the lesions ulcerate the carcinomatous tissue is found in the borders. The borders are rounded and nodular, hard, waxy, and of yellowish to pinkish color.

The base of an epithelioma presents the same characteristics of epitheliomatous tissue that are seen in the border. It is uneven and irregular, and made up of firm, irregular tubercles, here bright red rather than yellowish and waxy. In the most superficial lesions the indurated nodular base may be almost lacking because of the thinness of the layer of epitheliomatous tissue.

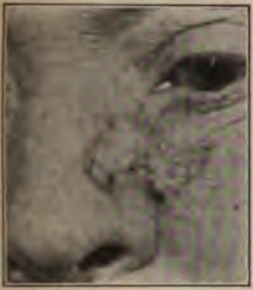


FIG. 293.—RODENT ULCER. (Author's collection.)

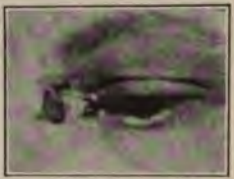


FIG. 294.—RODENT ULCER. (Author's collection.)

In all cases the lesions are very vascular. In the intact nodules dilated blood vessels can be seen over their surfaces, and in the ulcerating lesions dilated blood vessels can always be found in their border. These dilated blood vessels are of course an expression of functional hypertrophy produced by the demand of rapidly growing pathological tissues for an increased blood supply. The lesions always bleed freely. If they are ulcerating they are apt to bleed readily and freely upon slight causes.

The discharge from ulcerating epitheliomata is a viscid, scant secretion which dries into yellowish, varnishlike crusts. From secondary infection, that is always present in ulcerating lesions, there is more or less pus, which may be abundant and accumulate in thick, dirty, brownish or yellowish crusts, but ordinarily the purulent secretion is not abundant. In all forms of epithelioma a certain amount of deceptive healing may occur. Superficial lesions may heal over completely under favorable conditions, like careful dressing, and even in more destructive lesions enough connective tissue may at times be formed to produce scars, but in such healing nodules of epithelioma are left and sooner or later the disease pursues its usual course. In very rare cases the lesions disappear completely. This is in all probability in all cases due to an inflammatory process sufficiently intense to destroy entirely the carcinoma cells.

The pain accompanying epitheliomata, so long as they are confined to the skin, is usually not severe. Lesions may remain painless for years. It is only when they involve the deeper structures that they become painful. When that occurs, the pain usually becomes harassing.

Superficial epithelioma frequently begins as one or several confluent waxy tubercles which form either a small rounded or an irregular, nodular lesion. Over such a lesion the skin may remain shiny and smooth, or the surface may become scaly, with bleeding when the scales are removed.

Such a lesion may remain for months or years without ulceration, growing very slowly. At other times the lesion begins as a superficial erosion, or as a warty papule with an indurated base, or as a scaly patch. This scaly patch is most frequently a senile keratosis with a reddened base, and with a few dilated capillaries around it. Any of these beginning lesions will develop into a characteristic epitheliomatous ulcer: with rounded, more or less elevated, irregular borders, of round, discoid, or irregular shape, and with the ulcerated area showing a precipitate inner border and a nodular, infiltrated, red base. Such an ulcer at the beginning may be smaller than a split pea. Ordinarily it is from a quarter to half an inch or more in diameter. Being superficial it is movable. Its spread may be slow, but its tendency is persistently to increase, destroying in its course any tissue in its way.

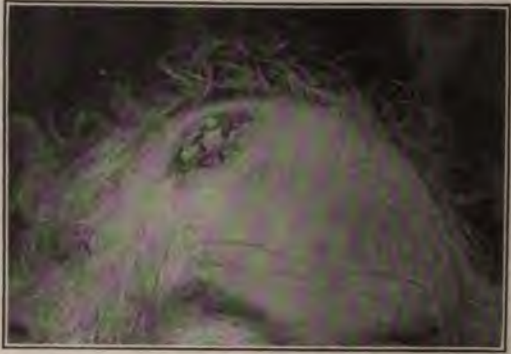


Fig. 295.—Deep Crateriform Epithelioma. (Author's collection.)

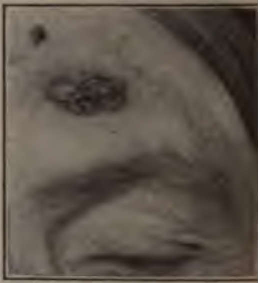
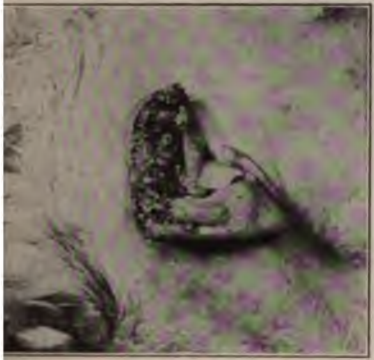


Fig. 296.—Small Nodular Epithelioma. (Author's collection.)

Rodent Ulcer (Synonyms: *Jacob's Ulcer, Noli me tangere, Tubular epithelioma, Carcinoma baso-cellulare, Krompecher's tumor*).—In some cases the spread of the epithelial proliferation is in a very thin layer in the corium, and the lesion which results is of very superficial type. The border is little, if at all, elevated, and shows least well the characteristic features of carcinoma. But even here, on close inspection, the border is still seen to be hard, rolled and indurated, and slightly nodular. The ulcers are at first superficial, round, or polycyclic, and spread slowly. Healing in these superficial lesions is not uncommon, and healthy scar tissue may at times replace a considerable part of the lesions. At times the lesions may heal over completely, but when this occurs borders or discrete tubercles of characteristic epithelioma will be found in the involved areas. These lesions show little tendency to deep involvement, but they may spread to an indefinite extent over the surface. I have seen such a rodent ulcer involve an area of more than a square foot on the back. Usually, sooner or later, in some part of such a lesion,

r involvement occurs with the production of a characteristic epi-
matous ulcer. The tendency to metastases in rodent ulcer is
t nil, but it does sometimes occur. Rodent ulcer is frequently,
ally by English authorities, described as distinct from superficial



. 297.—Epithelioma of Auricle. (Author's collection.)

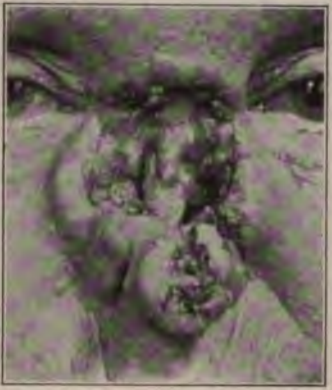
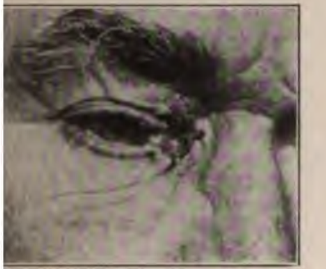


Fig. 298.—Deep Epithelioma of Nose. (Author's collection.)

lioma. Its superficial spread and the absence of metastases are
ar clinical features, but aside from these its clinical characteristics
stinctly those of epithelioma, and it is in my opinion neither advan-
s nor logical to erect any essential distinction between it and other
of epithelioma. This is the position of most continental
rities.



9.—Epithelioma of Inner Can-
hus. (Author's collection.)

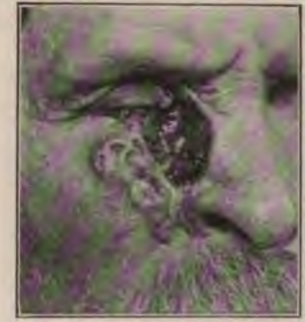


Fig. 300.—Deep Rapidly Growing Epithelioma. (Author's collection.)

poma-like Epithelioma.—In rare cases, the tubular type of epithe-
develops hypertrophic masses closely resembling lipomata (Fig.
These tumors have a pseudo-lobulation, are quite as soft as lipoma-
t usually have upon their summits a characteristic epitheliomatous
The microscopical picture is the usual one of basal cell epithelioma.

Morphœa-like Epithelioma.—Hartzell has called attention to a type of epithelioma infiltrating the skin in a way which gives it a resemblance to a patch of circumscribed scleroderma. The term describes very well certain rare epitheliomas which occur in flat patches of infiltration of a white or yellowish color. One such case in my experience showed at one part of the morphœa-like infiltration a dense collection of pigment. There may be a red and infiltrated areola, and telangiectases around and in the infiltration. While the name is a happy one as describing the peculiar appearance of the epitheliomatous infiltration, the condition itself, upon close examination, is not likely to be confused with morphœa.

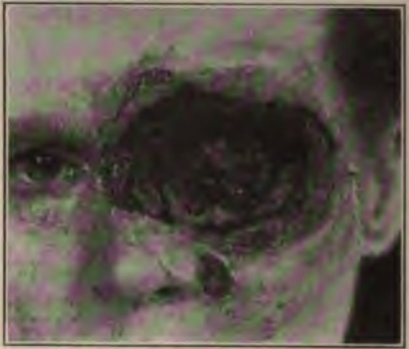


Fig. 301.—Epithelioma of Orbit; Late Stage. (Author's collection.)

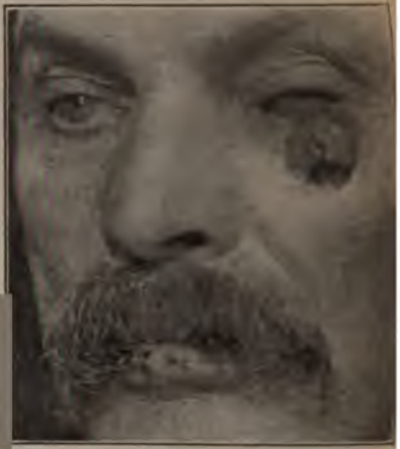


Fig. 302.—Superficial Epithelioma of Lip; Superficial Hypertrophic Epithelioma of Lower Lid. (Author's collection.)

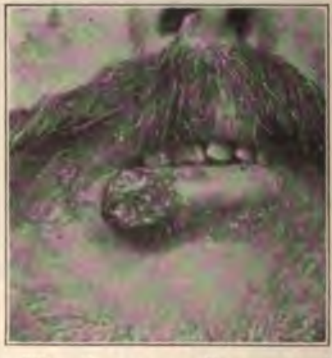


Fig. 303.—Superficial Exuberant Epithelioma of Lower Lip. (Author's collection.)

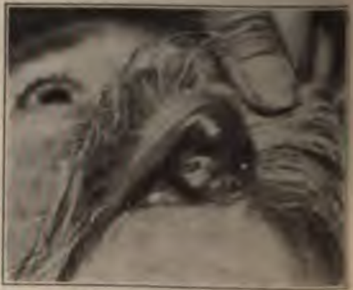


Fig. 304.—Rapidly Growing Nodular Epithelioma; Inner Surface Upper Lip. (Author's collection.)

Deep epithelioma begins as a single or a confluent group of subcutaneous nodules. The lesion at first is of pea to hazelnut size, and may be either a rounded or a conical projecting tumor, or show no elevation. It is hard to the touch, can be rolled under or between the fingers, and sometimes has a distinct crackling feel like a small onion or a minute Brussels sprout. The skin over it may not at first be involved; soon the skin is invaded, becomes of shiny red color, and the characteristic appearances of epitheliomatous tissue in the skin develop after this. Central ulceration quickly occurs. In these cases the ulcers formed are deeper than those seen at first in the other forms of epithelioma, and the amount of neoplastic infiltration around the lesions is greater than in the other forms. The borders and the base present the characteristics of other types of epithelioma.

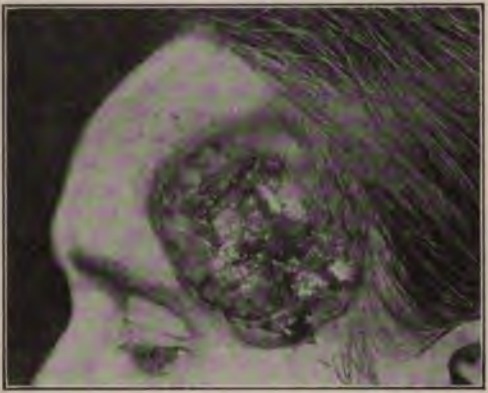


FIG. 305.—HYPERTROPHIC EPITHELIOMA. (Author's collection.)

Crater-form Epithelioma.—In many epitheliomatous ulcers the border, while rolled and nodular, is little elevated; frequently, however, an epithelioma develops as a large, projecting, conical nodule which ulcerates at its center with the formation of a sharply excavated, deep, central ulcer. Such lesions have very aptly been described by Hutchinson as *crater-form epitheliomata.* These may be superficial epitheliomata, but are more frequently deep-seated.

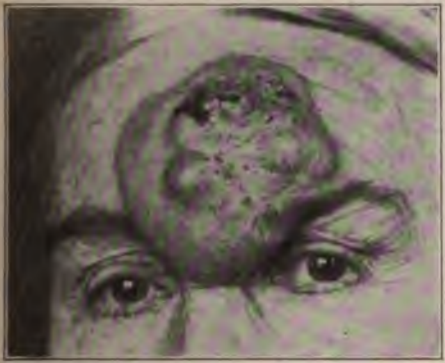


FIG. 306.—HYPERTROPHIC TUMOR COMPOSED OF SOFT EPITHELIOMATOUS TISSUE. Very unusual type of epithelioma. (Author's collection.)

Epitheliomata usually occur singly, but they are often multiple. They are most frequently multiple in cases of advanced senile keratosis where the epitheliomata develop from keratoses. In these cases most of the epitheliomata are small, inactive lesions, while only one or a few are actively growing and ulcerating.

Epitheliomata are most frequently found on the face and neck and about the muco-cutaneous junctures. Their most frequent location, according to most statistics, is the lower lip. The next most frequent location is upon the side of the nose or the cheek just below the inner

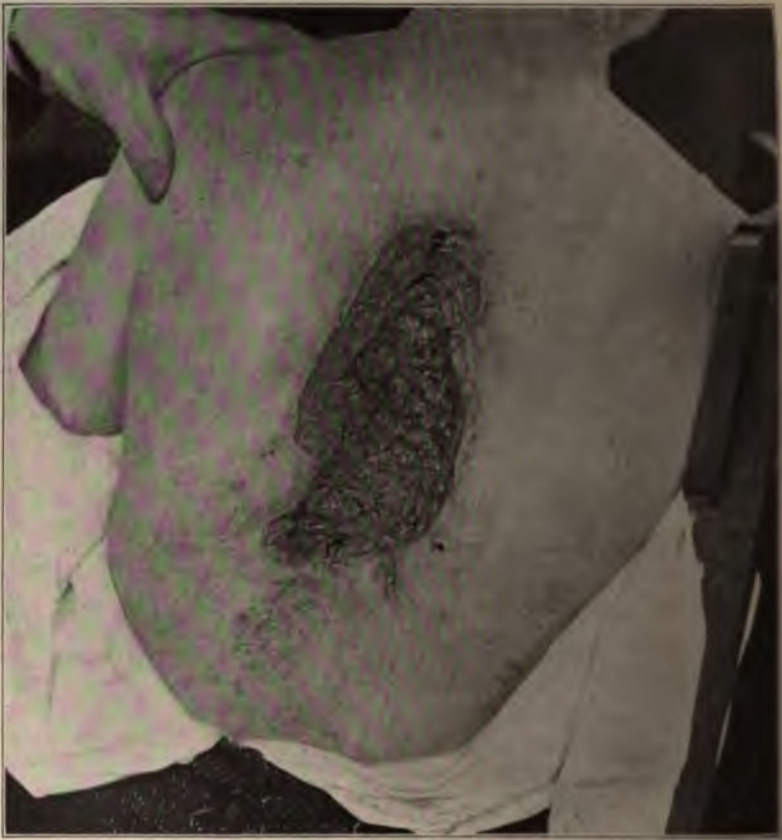


FIG. 307.—EPITHELIOMA OF BACK; BEGAN IN PSORIASIS PATCH. (Author's collection.)

canthus—where rodent ulcer usually develops—and they frequently develop upon other parts of the face. They are also common upon other parts of the face and upon the neck, scalp, and, to a less extent, the hands. They frequently occur also within the mouth, especially on the tongue, on the buccal mucous membrane at points exposed to irritation from bad teeth, and on the gums around bad teeth. They also occur, but less frequently, at the muco-cutaneous junctures of the genitals and anus.

There are certain factors which account for the distribution of epitheliomata. In the first place, they are liable to occur at points exposed to long irritation. They also show predilection for the muco-cutaneous junctures; this is probably due to the excessive irritation to which these junctures are exposed. The existence at given points of other lesions of the skin, especially if they are exposed to irritation, frequently accounts for the development of epitheliomata. This is seen in their development in scars, warts, and especially in keratoses, which are usually senile keratoses or keratotic patches on the mucous membranes known as leukoplakia. Indeed, most epitheliomata on the face and hands, excepting those on the lip, have their beginning in senile keratoses, and of those on the mucous membranes many begin in patches of leukoplakia. The development of senile keratoses into epitheliomata is a gradual process in which no abrupt point of transition can be found, and in the development of epitheliomata of this sort I am strongly of

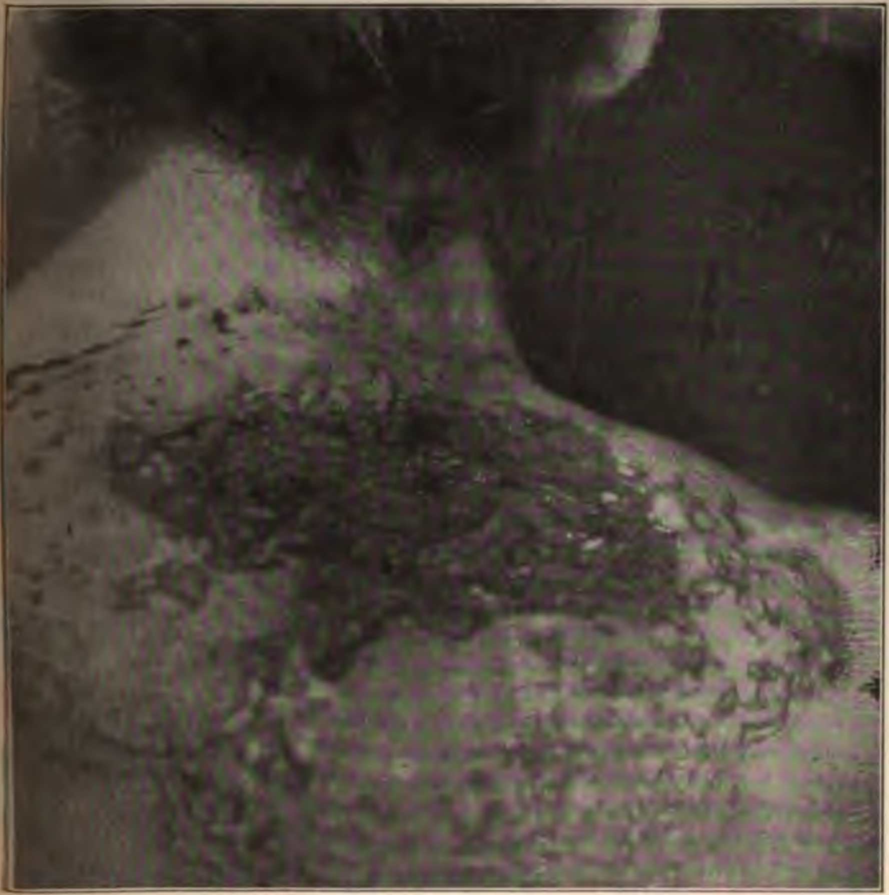


Fig. 308.—Very Large Rodent Ulcer of Back. (Author's collection.)

the opinion that the influence of the exposure of the parts to the elements is a most important factor, the effect of light on the parts being, I believe, the exciting agent. It is at least a fact that most epitheliomata occur upon those parts which are habitually exposed to light.

The rapidity of growth of epitheliomata varies greatly. Often small, superficial lesions remain quiescent for months or years, and perhaps never take on rapid growth. Other apparently similar lesions grow rapidly from the start, or may be inactive for an indefinite time and then take on rapid growth. In deep-seated epitheliomata the growth is usually rapid. There is almost no limit to the extent of growth of an epithelioma. The ordinary epithelioma in many cases never produces a lesion larger than a coin. Sooner or later, however, many epitheliomata if not destroyed involve the deeper structures, and then they gradually destroy all tissues which they meet. Beginning as a lesion, on the cheek for example, which may remain smaller than a finger nail for years, an epithelioma may finally grow rapidly and destroy the entire side of the face. Or beginning as a small, deep-seated nodule, on the lip for example, it may rapidly involve the surrounding structures and the contiguous glands until in a few months the lower jaw, the floor of the mouth, and the tissues of the neck are riddled.

The extent of spread which is possible in rodent ulcer has already been referred to. As it occurs most frequently on the cheek, near the eye, the greatest destruction which it produces is usually about the eye and in the orbit. The danger of metastasis in epithelioma varies chiefly with the depth of the lesions, but also to a degree with its location. Superficial epitheliomata, which proliferate outward forming exuberant growths, or which spread along through the corium without extending deeply beneath it, are least liable to have metastases. This is especially illustrated in rodent ulcer where metastases almost never occur. On the other hand, epitheliomata, which develop beneath the body of the corium and which accordingly grow under pressure, are very likely to have metastases form quickly. Metastases are quick to form from epitheliomata which are situated upon surfaces richly supplied with lymphatics and in intimate connection with near-by nodes. Thus lesions on the tongue are almost invariably accompanied by the development of carcinoma in the neighboring glands. The danger is a little less imminent when the lesions are on other parts of the mucous membrane of the mouth. At the muco-cutaneous junctures, like the lips, the dangers of metastasis are also very great; but even upon the lips, if the lesions are superficial and slow-growing, metastases may be very slow to develop. The same statements apply to lesions about the genitals and the anus. Upon the skin at points not in contact with the muco-cutaneous junctures, the danger of metastases is less. In superficial lesions the growths may extend very widely without metastases occurring. Whenever the lesions become deep, especially if they are growing under pressure, the liability of metastasis becomes greatly increased.

Etiology.—There are several well-determined exciting factors in the etiology of epithelioma. Among these are age, heredity, long-continued irritation, traumatism, and the presence of other persistent lesions of the skin. As in other forms of carcinoma the tendency to epithelioma increases with age. Most cases occur after forty, and epitheliomata are very common lesions in those sufficiently old to show well-marked senile changes in the skin. They are rare under thirty, but cases in the young are by no means unique. I have had several cases between twenty-three and thirty. Hartzell has reported a case at fourteen, and even younger cases have been observed. Of course in xeroderma pigmentosum epitheliomata may occur in infancy or even be congenital. There is often a history of epithelioma in successive generations of a family, and there is little room for doubt that an hereditary tendency is a factor in its etiology, but this tendency consists solely, in my opinion, in a family predisposition to those senile changes in the skin in which epithelioma is likely to occur. The development of epithelioma can frequently be attributed directly to chronic irritation, especially if this irritation occurs upon an existing lesion in the skin. This influence is seen in the development of epitheliomata in the mouth at points irritated by rough teeth or ill-fitting plates. It is commonly seen in epithelioma of the penis, which is nearly invariably associated with phimosis. The influence of smoking in the production of epitheliomata of the lip, and in the production of leukoplakia on the mucous membranes, which is often followed by epithelioma, have long been noted, and there is no reason to doubt its importance. My experience, however, in epithelioma of the lower lip is such as to lead me to believe that the effect of smoking is exaggerated. I have not seen epithelioma in the lower lip of a woman, but it is surprising how frequently I have seen epithelioma of the lower lip in men who have never smoked. The proportion is certainly as large as the proportion of nonsmokers among my patients. A form of epithelioma strikingly illustrating the effect of irritation is the so-called *chimney sweep's cancer,* which occurs on the scrotum as the result of irritation of soot and dirt. It is seen almost exclusively among English chimney sweeps. A similar form of epithelioma, occurring on the scrotum and forearms, is found in workers in paraffin and coal tar, as the result of irritation produced by these substances. Occasionally epithelioma develops from a slight wound of the skin, or from a pathological process producing an abrasion of the epidermis, like a simple herpes. These cases nearly invariably occur in the old, and occur most frequently where the wound has been irritated, as, for example, from the application of nitrate of silver. While this beginning of epithelioma is not rare, it is not nearly so common as from long-continued slight irritation.

The development of epitheliomata from other lesions in the skin is a common occurrence. This happens most frequently in lesions which are characterized by hyperkeratosis. The typical lesions from which epitheliomata develop are senile keratoses and leukoplakia, but they are prone to occur in lesions of all sorts characterized by hyperkeratosis. They may develop in callosities, in horns, in warts, and they are common complications of the other forms of hyperkeratoses, as in x-ray keratoses and in the rare cases of arsenical keratosis. They also occasionally develop in lesions of psoriasis. At times epitheliomata develop in lesions which are essentially lesions of the connective tissue. They sometimes occur in chronic ulcers, like varicose ulcers of the leg. They may also develop in the scars of lupus, or syphilis, or indeed in any scars; and Fordyce has recorded a case in which epithelioma developed in a burn before healing of the ulcer occurred. The development of epitheliomata, however, is much less frequent in lesions of this sort than in lesions which are characterized chiefly by hyperkeratosis. Their development is sometimes seen in congenital lesions, like moles, and larger nævi, and multiple benign epitheliomata; here they may have their origin in misplaced embryonic epithelium.

Epithelioma is said to occur from two to five times as frequently in men as in women. Aside from epithelioma of the lower lip, which in my experience has occurred exclusively in men, epithelioma is equally common in my experience in men and in women.

Pathology.—The direct cause of epithelioma is not known. There are certain facts, such as the influence of light in the production of epitheliomata in xeroderma pigmentosum, and the influence of x-rays in the production of epitheliomata in chronic x-ray burns, which strongly suggest that the lesions resulting from these causes at least are the result of degenerative changes independent of any infection. It is possible that there may be a multiplicity of causes of epitheliomata and of other forms of carcinoma, and that some are the result of primary degenerative changes of the tissues, while others are due to organisms.

Epithelioma may originate in the rete mucosum, in any of the follicles of the skin, or in misplaced embryonic epithelium. It probably in most cases begins in the rete.

Epithelioma occurs in two forms: the TUBULAR and the LOBULAR. The tubular represents the superficial or rodent-ulcer type of epithelioma; the lobular the nodular type.

In the TUBULAR type the growth consists of a meshwork of strings of epithelial cells which extend along the lymphatic spaces of the corium.

These are really strings of epithelial cells, not hollow tubes, so that the term tubular is a misnomer. In this form of epithelioma the growth extends superficially rather than deeply, the explanation for which may possibly be that the lesions occur typically where the tissues are thin and where the lymphatics tend to pursue a superficial course parallel to the surface. It is characteristic of rodent ulcer that necrosis occurs *pari passu* with the lateral extension of the growth. The explanation of this would seem to be found in the facts that the epithelial proliferation infiltrates so completely the lymphatic spaces in the area involved, and the connective-tissue stroma between the epithelial masses is so thin, that there is no opportunity, through lack of nutrition, for the masses to attain any macroscopic bulk before necrosis occurs. In the tubular form of epithelioma the cells are of smaller size than in the lobular form, and unlike the cells of the lobular form, they show no tendency to undergo the metamorphosis of normal epidermal cells. The rate of proliferation also is slower than the tubular form of epithelioma.

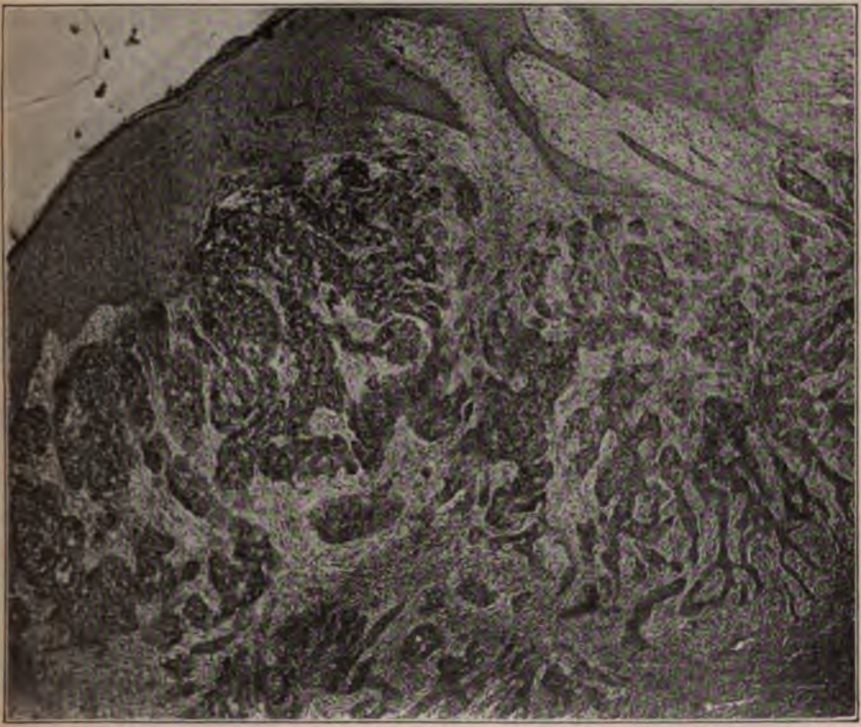


FIG. 309.—TUBULAR EPITHELIOMA. (Author's collection.)

Through the kindness of A. D. Bevan I have been able to study one section of epithelioma which was truly tubular. The growth was clinically a superficial epithelioma, and its highly malignant character is indicated by the abundant mitoses in the section. The epithelial proliferation extended through the tissues in the same way as in rodent ulcer. In some places they were solid cords, but in others they were hollow tubules presenting the structure of a cylindroma, in this respect being entitled to the designation malignant cylindroma.

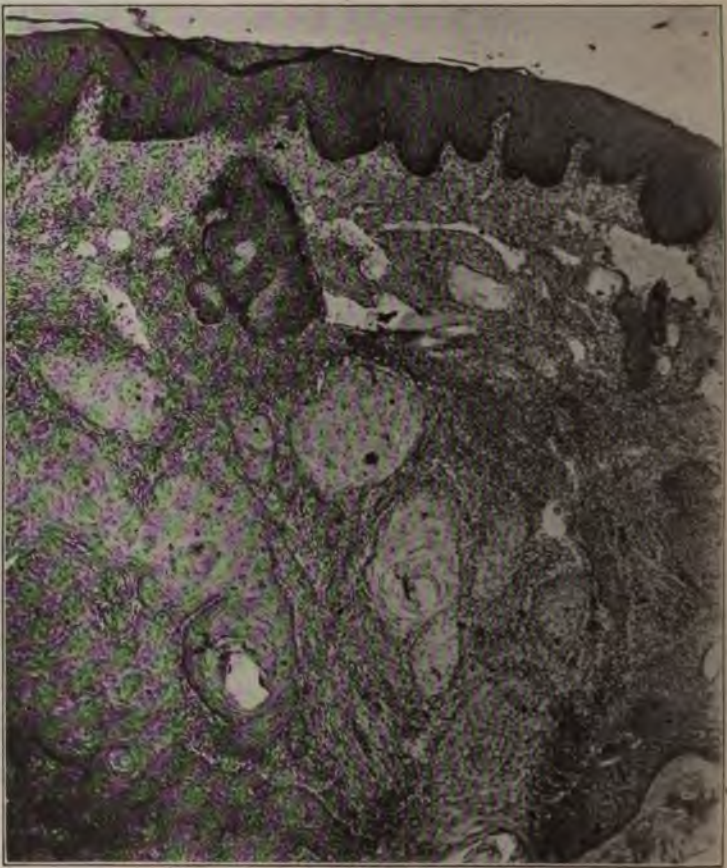


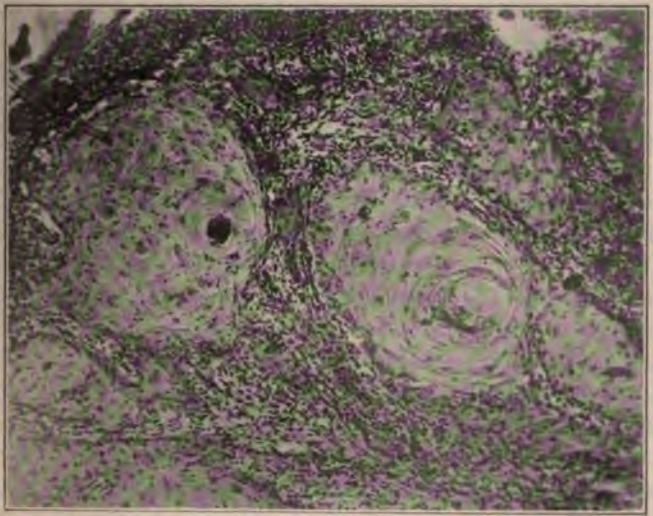
FIG. 310.—LOBULAR EPITHELIOMA. (Author's collection.)

The LOBULAR type of epithelioma consists of a downward and lateral growth of epithelial cells into the underlying connective tissue. The growths occur as large, fingerlike masses or lobules which branch and fuse with adjacent masses to inclose areas of connective tissue. The cells composing these masses may or may not show prickle formation. In this type the cells tend to undergo the evolution into horn cells which occurs in the normal epidermis, with the result that in some of the down-

ths there are formed the so-called epithelial pearls. These consist
ncentric masses of flattened cells which have a hyaline appearance
stain poorly. In all epitheliomata the connective-tissue stroma is
eat of chronic inflammatory process. In case of ulceration the
ficial parts of the growths show a more or less marked leucocytic
ration, due to secondary infection.

iagnosis.—The most characteristic clinical feature of epithelioma
e nodular, hard, waxy border. Next to this is the nodular character
e base. Other diagnostic features of greater or less importance are
ge of the patients, the tendency to free bleeding of the lesions, and
ndency of the lesions to develop at the muco-cutaneous junctures, at
s of irritation, and in other lesions, particularly scaly or horny
ns. The clinical features of epithelioma are in nearly all cases suf-
tly sharp to enable the expert to make a diagnosis upon them alone,
n account of the importance of the prognosis the safest course to
ue in nearly all cases is to confirm the clinical diagnosis by micro-
cal examination.

he most important matter in epithelioma from a practical stand-
t is to recognize the lesions early, when cure is easy. In old people



IG. 311.—EPITHELIAL PEARLS IN DEEP-SEATED EPITHELIOMA OF THE SKIN.
(Author's collection.)

ossibility of epithelioma should be remembered in all persistent
s, especially about the head and neck and hands, which are con-
ly inflamed or show evidence of degenerative changes. Warts,
, senile keratoses, fissures, and circumscribed lesions of any sort in

the old which show evidence of irritation or of growth should always be regarded with suspicion.

The two conditions which are constantly confused with epithelioma are lupus and tubercular syphilide, especially about the nose. Lupus should hardly ever be confused with epithelioma except in the unusual cases where epithelioma develops before forty, for lupus never develops in old age.

The clinical characteristics of ulcerating tubercular syphilide are sufficiently sharp to enable the expert to distinguish it from epithelioma, but in doubtful cases a section should be taken from the border for microscopic examination. It is important that the examination of the tissues be made by an expert pathologist, for the microscopical picture of a syphilide may closely simulate an epithelioma. In those cases in which any doubt remains between syphilide and epithelioma, if the removal of the epithelioma involves a mutilating operation, it may be desirable to test the case by specific treatment. If the lesion is an ulcerating syphilide, two weeks' specific treatment with surgical dressing should be sufficient to produce very marked improvement.

Prognosis.—The successful treatment of epithelioma involves the complete destruction of the carcinomatous tissue. The prognosis therefore varies according to the prospects for complete removal which are presented by any lesion. Epitheliomata which are confined to the skin can usually be cured. If they have involved bony structures the prospect of cure is less, and if there are glandular metastases the prospect is still less. Lesions, therefore, at the muco-cutaneous junctures and on the mucous membranes are the most unfavorable, because of the great danger of metastasis; while lesions upon the skin not closely in relation with lymphatic nodes offer the best prospect of radical cure. The rapidity of growth of a lesion is a factor of some importance. Rapidly growing lesions are less promising than indolent lesions, although rapidly growing lesions, if they can be gotten before metastases has occurred, can usually be cured. The depth also of the primary mass is a factor in the prognosis. If the mass is deep in the skin it is less promising of relief, both because the danger of metastases is greater and the rate of growth is more rapid.

Treatment.—The only radical treatment of epithelioma is complete destruction, and it is a principle applicable in all cases that irritating measures that do not cause complete destruction of the epitheliomatous tissue should not be employed. For that reason it is bad practice to apply superficial caustics like nitrate of silver or carbolic acid or corrosive sublimate. The irritation which they produce simply increases the blood supply and stimulates the growth of the tumor.

If an epithelioma is ulcerating it should be kept clean and dressed with bland applications according to the usual practice in treating ulcers. The best dressings for epitheliomata are wet dressings made with a non-irritating antiseptic or sterile solution, of which boric-acid solution is as good as any.

The radical treatment of epithelioma may be carried out by various destructive procedures, of which the ones usually employed are excision, curetting, electrolysis, the application of caustics, and exposure to x-rays.

Removal of epitheliomata by excision is the method usually employed by surgeons, and it is the only method which is justifiable in cases which demand removal of the contiguous lymphatic glands at the same time with the epithelioma. This principle applies especially to deep-seated epitheliomata of the lip, and no case of epithelioma of the lip should be treated by any other method than radical excision if removal of the lymphatics beneath the jaw is indicated and feasible. Aside from cases of this sort, excision is in my opinion not as good a method of treating epitheliomata upon the surface of the skin as either the application of x-rays or of caustics. The objection to excision of epitheliomata is that the outlying radicals of the tumor extend well beyond the apparent border of the growth, and their removal by excision requires the sacrifice of a great amount of healthy tissue. The deformity which results is therefore greater. But a more important objection is that even the most careful surgeon is apt to fail to get all traces of the growth, so that recurrence is common. From a large experience in epitheliomata, I am convinced that the danger of recurrence is greater in epitheliomata after excision than after removal by x-rays or caustics. Let me emphasize that I am speaking only of epitheliomata of the skin, and not of those on the mucous membranes or where removal of glands at the primary operation is indicated. In such cases I believe operation followed by x-rays is the ideal procedure.

In the treatment of epitheliomata by excision the lines of excision should extend well beyond the borders of the growth, at least a quarter of an inch or more beyond the apparently involved area, and care should be taken not to cut through the tumor tissue in making the dissection; otherwise particles of epithelioma are liable to be transplanted by the knife to healthy tissues and later show as recurrences.

Simple curetting is not a satisfactory method of treating epitheliomata, because all of the epitheliomatous tissue cannot be destroyed in this way; but combined with the subsequent use of caustics it is a good, quick method of treating small lesions. The friable epitheliomatous tissue breaks down readily under the curette, and macroscopic epitheliomatous masses can readily be scooped out. If this is done, the base should then be thoroughly cauterized with a strong caustic like a saturated solution of caustic potash or zinc chloride. This serves the double purpose of destroying chemically most of the remaining epitheliomatous tissue, and of producing a violent inflammatory reaction which will destroy any isolated cells. The procedure is rendered painless by the injection of a few drops of a two-per-cent or four-per-cent solution of cocaine or eucaine. Afterwards the lesion should be dressed with non-irritating wet dressings until the slough separates and the ulcer heals. Care should be taken to prevent the accumulation of pus crusts. Indeed, in the treatment of all epitheliomata one should never trust to healing under crusts, where the character of the granulations cannot be seen.

Small epitheliomata can be destroyed by electrolysis, which is simply a method of cauterization by caustics produced around the needle by electrical decomposition. The amount of destruction, however, produced around the needle is small and the method is not safe unless great care is taken to destroy the tissues thoroughly and widely. It is not a radical method and is not in my opinion to be commended.

The use of caustics, especially of caustic pastes, is not generally looked upon with favor. The reason for this I believe lies largely in the facts, first, that it is different from ordinary surgical procedures, and second, that it is used and exploited to a great extent by irregular practitioners.

The method has been advocated in this country notably by Robinson, and my experience with it enables me to indorse it fully. Like any other method of treatment it must be radical to be successful, and must be properly carried out. The great advantage of the use of caustics is that they produce a zone of violent inflammation around the mass which they destroy, so that they destroy not only the epitheliomatous tissue actually cauterized, but, through the inflammatory process excited, also outlying masses which have not been actually reached by the caustic.

In the use of a caustic, the application should be made vigorously enough to destroy immediately the tumor tissue which is apparent. The only exception to this is in the treatment of very large lesions, which have to be destroyed piecemeal by repeated treatments. Suppose, for example, we have an epithelioma the size of a ten-cent piece. The caustic should be so applied as to completely destroy the evident tumor. If this is done, inflammatory reaction may be relied upon to destroy the outlying radicals of the growth. If the lesion which we have assumed is a nodular lesion which is not ulcerating, it may be better to curette out the larger part of the mass and then apply the caustic. If it is an ulcerating lesion, the curette may be omitted and the tumor directly destroyed by the caustic. In small lesions of this sort caustic potash or chloride of zinc may be directly applied. They are most conveniently applied when they have been liquefied by the addition of just enough water to convert them into a sirupy liquid. The application can then be made either on a glass rod or on a cotton swab. Both of these caustics liquefy the tissues rapidly, and the destruction extends beyond the apparent limits, so that as soon as the entire tumor mass has been destroyed the action of the caustic should be stopped by washing the surface with water. The eschar formed should be treated in the same way as already suggested in considering curettement.

An equally satisfactory method of treating these small lesions, and a more satisfactory method for larger lesions, is by the use of caustic pastes. Of these the two most satisfactory pastes are Marsden's arsenious-acid paste and Bougard's chloride-of-zinc paste. In my experience both give equally satisfactory results. Marsden's paste consists of powdered acacia 1 part and arsenious acid 1 or 2 parts by weight, which are mixed at the time they are to be used with enough water to make a paste of the consistence of a firm ointment. If deep destruction of the tissue is necessary it is better to use the stronger mixture. If the lesion is relatively shallow in comparison with its area, the mixture of equal parts of arsenious acid and acacia are sufficient. In ulcerating lesions the stronger paste is preferable, because the eschar is formed more quickly and limits the absorption of arsenic. The paste is applied in a thick layer over the area to be destroyed, and should extend beyond the apparent border of diseased tissue. It should be left in position until a white eschar is produced; this requires from eight to twenty-four hours. If upon removal of the paste it is found that part of the tumor has not been destroyed, it should be reapplied immediately to destroy the remaining portion. After the eschar forms, it should be dressed surgically. Robinson says that no care should be taken to dress the wounds antiseptically, as suppuration increases the inflammatory process and renders it more destructive to remaining carcinomatous cells, but I have found no disadvantage in keeping the wounds clean. The slough will separate in from five to seven days, and if the treatment is properly carried out there remains a healthy granulating wound which heals in about a week and leaves a relatively small scar. The formula for Bougard's paste is:

℞ (1) Farinæ tritici (wheat flour), (2) Amyli,	āā	ʒi;
(3) Acid. arsenos. pulv		gr. viij;
(4) Hydrarg. sulph. rub		℈ij;
(5) Ammon. chloridi		℈ij;
(6) Hydrarg. bichlor. corros		gr. iv;
(7) Zinci chlorid. cryst		ʒi;
(8) Aquæ fervid		ʒiss.

The first six of these are separately powdered and then mixed together. The zinc chloride is dissolved in the boiling water, and this solution is added slowly to the other ingredients with constant stirring to prevent lumpiness. The resulting paste is firm and rather stiff. It has the advantage over Marsden's paste that if put in a tight jar to prevent evaporation it may be kept indefinitely. It is used in the same way as Marsden's paste.

Bougard's paste is slightly more destructive to normal tissue than arsenious-acid paste, but its application is less painful, and on the whole in my experience is the preferable paste, although Robinson prefers Marsden's. The application of Bougard's paste is rendered almost painless if five to ten per cent of eucaine is added to the water with which the paste is mixed. To Marsden's, Robinson adds ten to twenty per cent of cocaine or eucaine. In order to avoid all risk of arsenical poisoning, one should not use Marsden's paste over more than a square inch of surface at one time. Bougard's paste can safely be used over a larger area, but in order to keep perfect control of the destructive process it is better not to use it over more than a square inch of surface at one time. Much larger areas can be treated successfully by successive applications from day to day over different parts of the lesions. With both Marsden's and Bougard's pastes, if the epidermis is unbroken it should be removed with caustic-potash solution, as the pastes will not act through the horny epidermis.

The application of x-rays in the treatment of epithelioma depends upon the fact that by x-rays one is able to cause degeneration and ultimate destruction of carcinoma cells, while there is produced in the connective-tissue stroma only a proliferative inflammatory process which results in the formation of healthy scar tissue.

In the treatment of epithelioma with x-rays, the surrounding surfaces should be protected by a lead mask at least one fiftieth of an inch thick, in which a hole is made corresponding to the area to be exposed. This should be large enough to allow the exposure of the apparently healthy skin for about a quarter to half an inch beyond the border. The first aim in giving the exposures should be to produce a moderate x-ray reaction. To this end exposures should be given at intervals of from one to three or four days, and should be continued until involution of the tumor begins. In many cases this begins before any evidence of x-ray effect upon the skin occurs. Frequently epitheliomata entirely disappear, and are replaced by healthy scars without the production of any manifest effect upon the skin. Usually it is necessary to carry the exposures to the point of producing a moderate erythema. Only rarely is it necessary to push the exposures to the point of producing a vigorous dermatitis in order to get involution of the tumor. In my practice I give exposures of from five to fifteen minutes' duration with the wall of the tube at a distance of from two to six inches from the lesion, and repeat these exposures from two to six times weekly. With such frequent and close xposures a relatively small amount of x-rays should be used, just enough o cause a yellowish-green glow in the tube. If stronger exposures are iven they should be correspondingly shortened in time, or given less requently. In cases where there are unusual reasons for haste, as, for xample, with deep, rapidly growing epitheliomata, I push the treatment from the start, and produce a dermatitis quickly. But except under hese conditions it is better, in my opinion, to treat lesions more slowly nd obtain the result gradually. In this way all risk of troublesome urns can be avoided, and in nearly all cases successful results can be ttained. To carry out the treatment from the beginning until a healthy ar is obtained usually requires from three to ten weeks. In many cases he involution of the tumor can be started by from four to six exposures, r less if they are given vigorously. When involution begins, or x-ray ythema appears, the exposures are stopped, and, as a rule, it requires rom two to three weeks for the lesion to entirely disappear. Occasionly it is necessary to repeat the treatment.

The chief advantage of the treatment of epitheliomata by x-rays is hat the agent can be made to destroy diseased tissues without destroying he healthy connective-tissue stroma; the method, therefore, leaves small ars, and further is applicable to many lesions which are so extensive r involve such important structures that removal is impossible. Minor dvantages are that the treatment is painless and avoids more radical rocedures. Roentgentherapy is also a very valuable means of retarding he growth of inoperable tumors, even where complete destruction is mpossible. In my experience x-rays are applicable to all forms of epihelioma, and on account of its advantages it is the preferable method of reatment, as a rule, except in the type of cases requiring a radical operaion, which have been referred to in considering treatment by excision. n my experience in the treatment of epitheliomata with x-rays, extendng over six years, the results are as a rule permanent, with certainly as ew recurrences as take place after any other method of treatment.

III.—PAGET'S DISEASE OF THE NIPPLE[1]

SYNONYM: *Malignant Papillary Dermatitis* (Thin).

Paget's disease is a disease usually developing in the nipple and reola, which is characterized first by a sharply circumscribed area of ermatitis, but later shows malignant proliferation of the epithelium

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[1] Paget's disease of the *nipple* is a misnomer, as the disease occurs elsewhere bedes the nipple, but inasmuch as osteitis deformans is also known as Paget's disease, he title of Paget's disease alone for the skin affection is open to possible confusion.

Paget, *St. Bartholomew's Hospital Reps.*, 1874, vol. x, p. 87.—Duhring and Wile, *mer. Jour. Med. Sci.*, vol. lxxxviii, 1884, p. 141.—Wickham, "Maladie de la peau and the clinical features of epithelioma. Clinically Paget's disease may be described as a sharply circumscribed area of dermatitis which subsequently becomes a superficial epithelioma, and, if situated on the nipple, is usually followed by carcinoma of the breast.

Symptomatology.—The disease begins with scaliness and slight thickening of the epidermis of the nipple and areola, with a tendency to fissures and excoriations. Gradually this becomes converted into an area of dermatitis, red, weeping, and showing a finely granular surface. The discharge is yellowish and viscid, and dries upon the surface into varnishlike crusts. Unlike ordinary eczema the border of the inflamed area is sharply circumscribed, and there is a superficial but firm induration of the area so that when grasped between the fingers it feels like a rounded disk embedded in the skin. Gradually the infiltration becomes somewhat greater; the borders become slightly elevated, and the nipple melts away. Partial healing may take place upon the surface, and the lesion then shows a striking resemblance to a superficial, nonulcerating epithelioma. When the breast becomes involved, there may be retraction at the nipple site, and characteristic nodular infiltrations are to be felt in the breast.

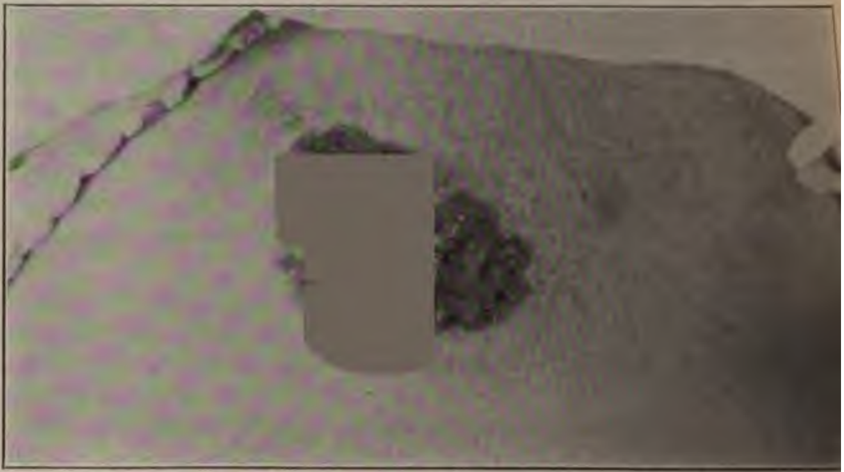
FIG. 312.—PAGET'S DISEASE OF THE NIPPLE. (Hartzell's collection.)

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dite maladie de Paget," *Thèse de Paris*, 1890, *Annales*, pp. 45 and 139 (bibliography).—Elliot, *Jour. Cutan. Dis.*, 1892, p. 272.—Jackson, *Jour. Cutan Dis.*, 1896, p. 428 (bibliography).—Jungmann and Pollitzer, *Dermatolog. Zeitschr.*, June, 1904.—Fox and MacLeod, *Brit. Jour. Derm.*, 1904, p. 43.—Fordyce, *Jour. Cutan. Dis.*, 1905, p. 193.—Sequeira, *Brit. Jour. Derm.*, December, 1905, p. 454.—Fowler, *Jour. Cutan. Dis.*, January, 1906, p. 27.—Hartzell, *Jour. Cutan. Dis.*, July, 1906.

The disease in nearly all cases begins in one nipple, for some reason more frequently on the right side, but it may develop later on the other. Cases have been observed on the scrotum, buttocks, umbilical region, and elsewhere. As a rule, on the breast it is confined to the nipple and areola, or spreads slightly beyond this area, but Jamieson and Elliot have recorded cases which spread over the entire surface of the breast and even into the axilla. The course of the disease is usually slow. Carcinoma of the breast may develop within one or two years, or, on the other hand, it may be postponed for ten (Duhring) or even twenty years (Jamieson). Like most superficial carcinomata, its spread over the surface is usually very slow. In its early stage of dermatitis there is some itching, or prickling, and at times radiating pains. Its later course is not characterized usually by pain, except when the pain of deep-seated carcinoma comes on.

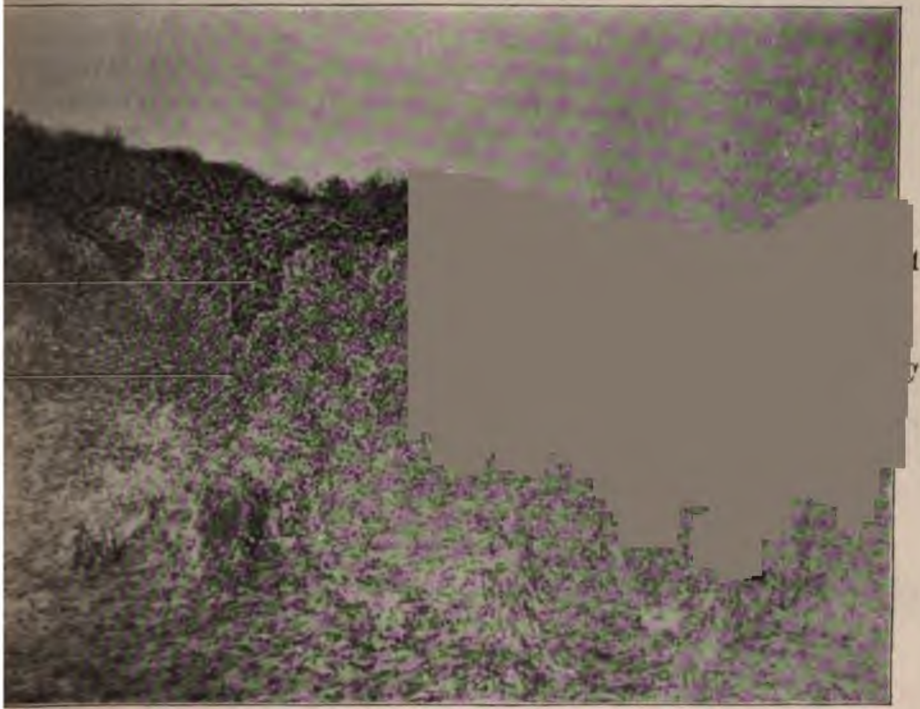

FIG. 313.—SECTION OF AREOLA OF THE BREAST. *A*, Ulcerating surface—the rete mucosum has been reduced to a mere remnant; *B*, down-growing proliferating epithelium; *C*, *C*, dense round-celled infiltration in the corium. (Hartzell.)

Etiology and Pathology.—On the breast it occurs in women of the cancerous age—after forty. Its occurrence in other localities than the nipple, in cases where irritation has apparently been a factor, suggests that long-continued irritation is an important factor in its causation. Indeed, it is not definitely settled that the disease in its inception on the nipple is not a simple dermatitis, and through long persistence develops epithelioma.

Paget's disease begins as an hypertrophy of the rete which sends down upon the corium clublike growths that compress and obliterate the papillæ. These proliferating downgrowths ultimately penetrate the basement membrane and produce epitheliomatous infiltration in the lymphatic spaces of the corium. The proliferating rete cells also grow along the lactiferous ducts and ultimately break through their walls with the production of carcinoma of the breast.

The characteristic feature in the histology of the disease is the peculiar degeneration which the rete cells undergo with the formation of the psorospermlike bodies first described by Darier. These are a diagnostic feature of the disease which distinguishes it from other forms of epithelioma. They were formerly thought to be coccidia and probably the cause of the disease, but they are now universally regarded as degeneration products. Microscopically they appear as double-contoured, highly refractile, round or oval bodies, usually containing one or more masses of chromatin in a clear space formed by the shrinking of the cell protoplasm. They are several times the size of the surrounding epithelial cells.

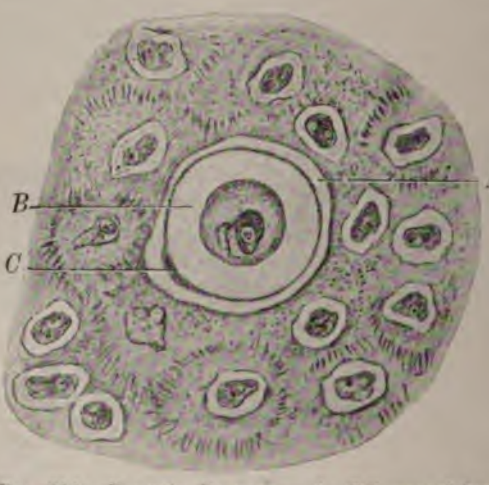

FIG. 314.—PAGET'S DISEASE. *A*, Cell containing *B*, a so-called psorosperm; *C*, nucleus of invaded cell crowded to one side by the parasite. (Darier.)

Diagnosis.—In the early stages Paget's disease of the nipple is to be diagnosticated from eczema of the nipple. Eczema of the nipple usually occurs during the period of nursing, is often upon both nipples, and occurs as a superficial dermatitis with an ill-defined, fading border, and without the disklike induration of Paget's disease, and it may be cured by the bland method of treatment effective in eczema. In eczema the psorospermlike bodies are absent. Except when Paget's disease occurs as a characteristic papillary dermatitis, the only positive way of making a diagnosis between it and other forms of superficial epithelioma is by demonstrating the psorospermlike bodies.

Prognosis and Treatment.—Sooner or later, if left alone, Paget's disease is followed by carcinoma of the breast.

Paget's disease before it involves the breast may be successfully treated by superficial caustics or x-rays. Several cases treated with x-rays have been reported. I have treated two cases successfully with x-rays, one of which has remained well for three years. Treatment with x-rays should not be undertaken if any suspicious nodules exist in the breast. In that event, the only proper treatment is prompt, radical removal of the breast.

IV.—PRIMARY PIGMENTED CARCINOMA OF THE SKIN[1]

Unna, Gilchrist, Johnston, and others have shown that some of the pigmented, malignant growths of the skin which have hitherto been regarded as sarcomata are pigmented carcinomata. The lesions usually begin in moles or larger pigmented nævi, and occur as pigmented growths which may be multiple or single. Their course is less malignant, as a rule, than that of pigmented sarcoma of the skin, but otherwise they cannot be readily differentiated from pigmented sarcoma. The diagnosis can only be made from the histological structure.

V.—SARCOMA CUTIS[2]

Sarcoma of the skin may be either primary or secondary. Primary sarcoma of the skin may occur as single or multiple tumors. Secondary sarcoma of the skin usually occurs as multiple tumors; its lesions in the skin are indistinguishable from those of multiple sarcoma of the skin.

Symptomatology.—All of the histological forms of both nonpigmented and pigmented sarcoma may occur in the skin, and, depending upon variations in texture and composition of the growths and on other variable factors, the clinical pictures which the disease in its different forms may present are exceedingly variable. The malignant character of the growths, however, is usually marked, and, as they differ distinctly in their clinical features from all forms of carcinoma of the skin except pigmented carcinoma, the diagnosis can readily be made by exclusion. The differential diagnosis from melanotic carcinoma of the skin is made only by microscopical examination.

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[1] Gilchrist, *Jour. Cutan. Dis.*, 1899, p. 117 (histology and bibliography).

[2] Hartzell, "Sarcoma Cutis," *Jour. Cutan. Dis.*, 1893, p. 21.—Bowen, "Mycosis Fungoides and Sarcomatosis," *Jour. Cutan. Dis.*, 1897, p. 65.—Gilchrist, "Melanotic Sarcoma," *Trans. Amer. Derm. Assn.*, 1898.—Schalek, "Melanosarcoma Cutis," *Jour. Cutan. Dis.*, 1900, p. 147.—J. C. Johnston, "Sarcoma and Sarcoid Growths," *Jour. Cutan. Dis.*, 1901, p. 305 (review and histology).—Lieberthal, *Jour. Amer. Med. Assn.*, December, 1902, p. 1454.—Wende, *Jour. Cutan. Dis.*, June, 1903.—Wilson and Kalteyer, *Amer. Jour. Med. Sci.*, November, 1903.—Johnston, "Melanoma," *Jour. Cutan. Dis.*, January and February, 1905 (complete review).

In spite of their diverse characters, there are still clinical features which sarcomata in the skin have in common. The growths are usually uniform, rounded or oval, at least in the start, without irregular nodulations of their surface. The epidermis over them, as a rule, is uninvolved and is smooth and shiny, or in some cases scaly and thickened. If they begin in moles the surface may be papillomatous and warty. If they are subcutaneous there may be no change in color, but if they involve the corium they are usually of a dark shade of red or even purple to black in color, and frequently they are markedly pigmented. Occasionally they become fungoid or ulcerative, but this is a late tendency and is not common as in carcinoma. Ordinarily the lesions are soft. The most vascular ones may even be pulsatile, but occasionally they are hard, fibroid growths. As a rule they are of more malignant character than carcinoma of the skin. They may remain quiescent for a long time, but when they become active their growth is rapid and metastases are likely to occur quickly.

Clinically they may be divided into ***nonpigmented sarcoma*** of the skin, which may be ***single*** or ***multiple***, and ***pigmented sarcoma*** of the skin. These are true sarcomata. In addition there are the so-called sarcoid growths, viz.:

Multiple idiopathic hæmorrhagic sarcoma of Kaposi,
Endothelioma or *sarcoma capitis,*
Multiple benign sarcoid of Boeck,
Mortimer's malady,
Leukæmia and *pseudoleukæmia cutis,* and
Mycosis fungoides.

The true character of this group is not certain.

Single or Circumscribed Nonpigmented Sarcoma.—This may appear as a tumor in the skin or in the subcutaneous tissue. If in the subcutaneous tissue, the lesion may be of normal or red color; if the body of the corium is involved, the lesion varies from red to dark-reddish brown or purple. The skin over it is usually smooth and glistening, but the epidermis may be cracked or scaly. The lesions are soft and very vascular, and abundantly supplied with dilated capillaries. They are likely to grow rapidly, up to the size of a hazelnut or an olive, or occasionally an egg, and they may become pedunculated or fungoid or ulcerative. At times they remain localized and grow slowly for months or a year or more, but sooner or later they are followed by metastases and general sarcomatosis, and death is likely to occur in from six months to

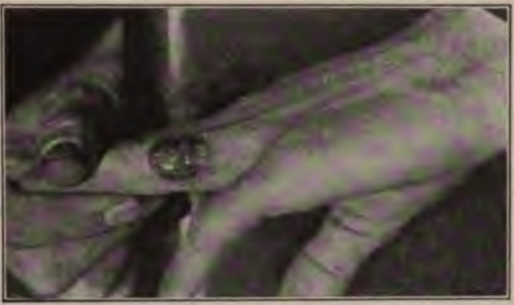

FIG. 315.—PRIMARY ROUND-CELLED SARCOMA OF FINGER. (Author's collection.)

years after the ap-
rance of the tumors.
e primary tumor may
followed by other tu-
rs of the skin, or, on
other hand, general
comatosis and death
y occur without other
ions developing in the
n.

Two cases of primary
npigmented sarcoma of
skin have occurred in my experience. Both were round-cell sarcom-
, and both curiously occurred on the hands; one on the thumb, one
the middle finger. One developed after a severe bruise, one after

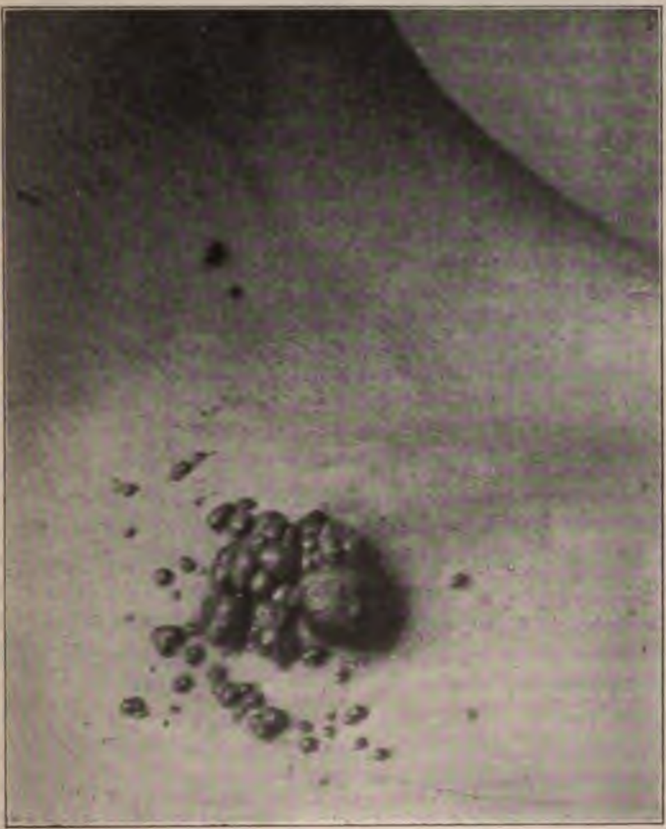

FIG. 316.—ANGIOSARCOMA. (Winfield's collection.)

injury caused by a ring. One patient was a man, aged twenty-eight, the other a woman over forty. In both cases the tumors were excised, and death occurred in the case of the man within twelve months. In the second case simple excision of the tumor was followed by persistent x-ray exposures, and the patient has remained well four years.

Multiple Nonpigmented Sarcoma of the Skin.—Nonpigmented sarcoma of the skin occurs in multiple tumors similar to the single type

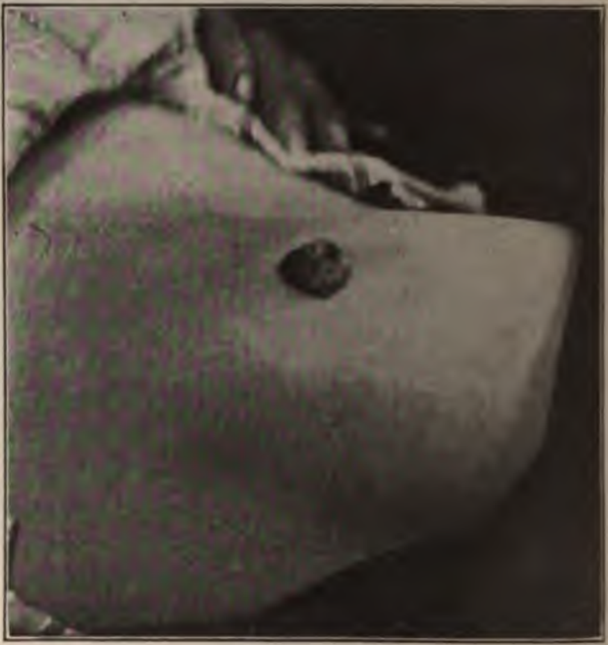

FIG. 317.—PIGMENTED SARCOMA BEGINNING IN A MOLE. (Author's collection)

above described. The tumors vary from the smallest pinhead lesions u to the size of a lemon. Their number may vary from a few to sever hundred lesions, and they are irregularly scattered over the body. Th tumors may be discrete and widely but irregularly distributed, or th may be grouped into confluent masses. This form of sarcomatosis ma be primary, or may be secondary to sarcoma in the viscera and oth tissues. The patients die from general sarcomatosis in from a few wee to one or two years.

Primary nonpigmented sarcoma of the skin is very rare. The t most definitely established factors in its etiology are injury and t existence of embryonic defects. The lesions may begin in apparen normal skin, especially after injury, but they usually begin in oth lesions, like warts, nævi, scars, or benign tumors such as sebaceous cy

Melanotic Sarcoma.—Primary melanotic sarcoma usually occurs as a single pigmented tumor. It begins in some pigmented structure and develops into a slaty, bluish, or blackish tumor. In such a case, the tumor may be a round, smooth, oval lesion from the size of a pea to that of an olive or larger, or it may be an irregular papillomatous, horny, blackened plate, such a lesion as one would expect from the enlargement of a black mole. Very rarely numerous lesions develop around the original growth before general sarcomatosis occurs. The disease is highly malignant, and metastases may occur very early in the growth of the lesion in the skin. In other cases, before metastasis occurs, the esions in the skin may grow to the size of an olive or larger, ulcerate, or remain stationary, or even undergo involution. They are usually followed by rapidly developing melanotic sarcomata in other tissues, and are fatal in from a few months to a year.

Melanotic sarcoma is the most frequent form of sarcoma in the skin. When primary it usually develops from pigmented moles, especially after rritation. Secondary lesions in the skin may result from a melanotic growth in any of the other structures of the body. The most frequent origin of secondary melanotic sarcoma in the skin is sarcoma in the pigmented structures of the eye.

MELANOTIC WHITLOW.—This name is given by Hutchinson to a form f pigmented sarcoma of the skin which begins as a pigmented spot at the order of the nail. It develops into a fungoid tumor and pursues the apid course of other melanotic sarcomata.

Pathology.—Sarcomata of the skin correspond to sarcomata in ther structures, and represent a new growth of embryonic connective ssue. Like sarcomata in general, they are prone to form metastases y way of the blood vessels, resulting in widespread secondary growths. They are composed of round, spindle, or giant cells, with a minimum mount of connective tissue or intercellular substance and a very rich lood supply. There is at times a formation of more or less distinct lveoli, probably due to the fact that the sarcoma cells grow into spaces f the normal connective tissue. The epidermis over the growths may e normal, or the sarcoma cells may grow into and destroy the pithelium.

Diagnosis.—A pigmented, growing tumor, beginning in a mole or ongenital deposit of pigment, is most likely to be a sarcoma; it may be a pigmented carcinoma, but the differential diagnosis can only be made by a microscopic examination. Too much emphasis cannot be aid upon the importance of recognizing the first evidences of beginning growth in a mole, for metastatic growths may develop from uch sarcomata before any marked change in the primary lesion has occurred.

Treatment.—Any circumscribed sarcoma of the skin, if operable, should be removed as soon as discovered. The excision should be made wide of the possible limits of the growth, and care should be taken to avoid transplantation of particles of the growths in the operation. The removal should be followed, in my opinion, by vigorous x-ray exposures carried up to the point of acute reaction, and this should be followed by subsequent but somewhat less vigorous exposures at intervals of a few months for a year or more. Various observers have noted the effect of x-rays on sarcomata in general, and wherever they occur in the skin a vigorous effort should be made to produce all effect possible from x-ray exposures. At the same time, vigorous treatment should be undertaken with the hypodermic administration of arsenic. Köbner, Kaposi, Sherwell, Wende, and others have reported cures from the use of arsenic hypodermically. The arsenic can be given in the form of Fowler's solution, solution of sodium arsenite, or of sodium cacodylate and should be pushed vigorously. Crocker has had involution of tumors from the administration of salicin. As a last resource, injection of erysipelas and prodigiosus toxins may be tried. None of these measures are likely to be of any benefit in melanotic sarcoma, although Lassar has reported a cure of one melanotic lesion from arsenic.

VI.—IDIOPATHIC MULTIPLE HÆMORRHAGIC SARCOMA OF THE SKIN[1]

SYNONYMS: *Idiopathic Multiple Pigmented Sarcoma* (Kaposi).

The disease was first described under the title idiopathic multiple pigmented sarcoma of the skin by Kaposi, who observed twenty-five cases. The pigment, however, in the lesions is blood pigment from extravasations of blood. They are not, therefore, true pigmented sarcomata, and as they have not the highly malignant character of pigmented sarcoma, the term hæmorrhagic sarcoma is better.

Symptomatology.—This form of sarcoma occurs as numerous symmetrical, plum-colored to dark-purplish or black tumors. The lesions vary in size from a small pea up to a walnut or larger. They are usually oval or round, but they may occur as flattened, circumscribed infiltrations. The skin over them is tense and glistening, and the tumors rarely ulcerate, although a few of the largest lesions may ate late in the course of the disease. The lesions are ordi- y firm; but they are highly vascular, are accompanied often by d blood vessels, and occasionally are so vascular that they suggest mata.

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[1] Fordyce, *Jour. Cutan. Dis.*, 1891, p. 1.—Sherwell, *Amer. Jour. Med. Sci.*, October, 1892.—Brayton, *Indiana Med. Jour.*, November, 1893.—G. W. Wende, *Jour. Cutan. Dis.*, 1898, p. 205.—Koehler and Johnston, *Jour. Cutan. Dis.*, 1902, p. 5.—Sellei, *Archiv*, 1903, vol. lxvi, p. 1 (bibliography).—Weber and Daser, *Brit. Jour. Derm.*, 1905, p. 135.—MacLeod, *Brit. Jour. Derm.*, 1905, p. 173 (bibliography).—Halle, *Archiv*, vol. lxxii, p. 373, 1905 (review and bibliography).

he distribution of the lesions is markedly symmetrical, and they specially numerous on the extremities. On the legs the lesions may ie aggregated into large masses, with the œdema which is then iced there may be great enlarge- of the parts. Late in the course e disease they may appear upon aucous membranes. The number imors gradually increases by the opment of new lesions, which in iced cases may amount to hun- . There is some tendency for the is to undergo spontaneous involu- This is sometimes shown by cen- lepression in some of the lesions; lesions may disappear entirely, in very rare instances there has entire spontaneous disappearance daway). This form of sarcoma slowest in its progress, but death ly occurs from metastasis in other . In Kaposi's experience the aver- uration of life was five years, but ionally the patients may live for years without disturbance of the al health. In Jackson's case, the at was in good health twenty-one after the appearance of the dis- in Taylor's, twenty-four years ; in Brayton's, twenty-five years ; and in one case in my experi- which is illustrated here, the patient was in fair health at the of the photographs, seventeen years after the beginning of the se.

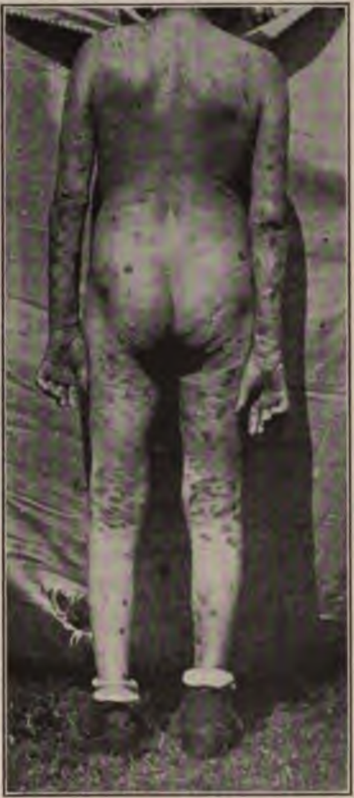

FIG. 318. — IDIOPATHIC MULTIPLE HÆMORRHAGIC SARCOMA. (Author's collection.)

tiology and Pathology.—Nearly all cases of Kaposi's type have in males of middle or old age. Nothing is known of its direct cause. iple hæmorrhagic sarcoma occurs as round or spindle-cell tumors ig a rich network of vascular sinuses and thin-walled blood vessels. iorrhagic areas exist throughout the growth in which there is a deposit of pigment derived from extravasated blood. By some authorities the disease is regarded as a granuloma.

Diagnosis.—Multiple hæmorrhagic sarcoma might be confused with mycosis fungoides, tubercular leprosy, or multiple syphilitic gummata, but aside from the histological differences the clinical differences are sufficiently sharp in these various infections to make a differential diagnosis. In mycosis fungoides there is a history of, and there are usually present, sharply circumscribed areas of itching dermatitis; the tumors are cauliflowerlike, bright red, and tend to ulcerate freely. In leprosy there are the characteristic facies, the presence often of bullæ, anæsthetic symptoms and other evidence of nerve involvement. Multiple gummata are never so numerous or so symmetrically distributed, and if abundant are ulcerative and show characteristic ulcers and scars.

Treatment.—The treatment hitherto of hæmorrhagic sarcoma has been unavailing. Arsenic has not been successful in Kaposi's hands, but should be tried. Halle[1] has seen great improvement from the exposures of the tumors to x-rays.

VII.—MULTIPLE BENIGN SARCOID[2]

Under this title Boeck has described a clinical picture very strikingly like that of leukæmia cutis. The histological structure of the growths, however, is unlike that of leukæmia cutis, nor does it conform to that of sarcoma. In a nodule examined by Pawloff there was a dense infiltration around the blood vessels, involving all of the corium and extending into the connective tissue. This was composed of connective-tissue cells with very few leucocytes or mast cells. In places fibroblasts were abundant, and at other points small islands of a uniform cloudy appearance existed, containing leucocytes and showing a degeneration resembling hyaline degeneration. Among the epitheloid cells were some large giant cells. The disease tends to disappear spontaneously, and usually disappears under the administration of arsenic.

VIII.—ENDOTHELIOMA CAPITIS[3]

SYNONYMS: *Sarcoma cutis, Turban Tumors, Cylindroma.*

This is a very rare form of connective-tissue tumor occurring on the scalp. It may occur as a lobulated group of tumors which vary in size from a nut to an apple, or may amount to a mass of tumors covering the scalp like a turban. The disease is slow-growing; it may persist for thirty or forty years and is not clinically malignant. Histologically the tumors are alveolar sarcomata or endotheliomata. In many of the cases there has been a family history of the tumors, and they are probably growths of congenital origin but of late development.

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[1] Halle, *Abs. Jour. Cutan. Dis.*, 1905, p. 415.

[2] Boeck, *Archiv*, vol. lxxiii, 1905, Nos. 2 and 3.

[3] Crocker, who refers to cases by Baker and Kaposi.—Cohn, *Jour. Cutan. Dis.*, 1893, p. 393.—Spiegler, *Archiv*, 1899, vol. l, p. 163 (complete review).

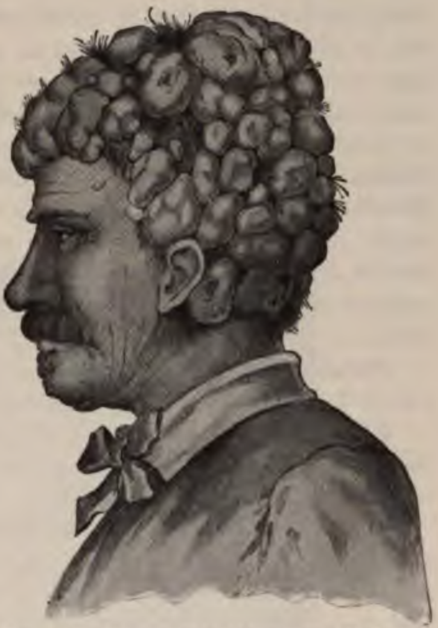

FIG. 319.—ENDOTHELIOMA CAPITIS. (Spiegler.)

IX.—MORTIMER'S MALADY[1]

Crocker included provisionally among the sarcoid growths a very rare affection which Hutchinson[1] has described upon the basis of four cases under the above name. The eruption occurs on the face and hands symmetrically in the form of raised, sharply defined, dusky red, flat tumors, which may persist indefinitely or undergo involution without ulceration, or ulcerate. It resembles somewhat Boeck's sarcoid, but its histology has not been studied.

X.—MULTIPLE SARCOID GROWTHS IN LEUKÆMIA AND PSEUDOLEUKÆMIA[2]

In leukæmia purpuric lesions in the skin are an occasional manifestation of the general hæmorrhagic tendency; and in both leukæmia and pseudoleukæmia pigmentations are not uncommon, probably as a result of the irritation produced by abdominal tumors. These lesions of leukæmia have been referred to already.

Leukæmia cutis.—In addition, a good many cases have been recorded in which sarcomalike growths have developed in the skin in leukæmia. The tumors occur in the skin or the subcutaneous tissue as small nodular tumors up to the size of a hazelnut, as larger tumors up to the size of an orange, and as patches of diffuse thickening and hypertrophy of the skin. The lesions vary from a few up to hundreds. Their color may be that of normal skin, or waxy, or they may be dark red or bluish, resembling multiple sarcoma of the skin. They may appear without preliminary changes in the skin, but a common beginning, which characterizes the type described by Kaposi as lymphoderma perniciosum, is in patches of scaling, moist dermatitis with intense itching. The lesions occasionally ulcerate, and in the ulcerative form the picture produced is much like that of mycosis fungoides. The lesions may appear anywhere upon any of the surfaces, but are apt to involve the face and extremities. On the face the diffuse lesions may produce leontiasis, with thickening of the skin and exaggeration of its folds.

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[1] Hutchinson, *Archiv of Surg.*, vol. ix. Plate CIII, p. 307.

[2] Linser, "Skin Changes in Pseudoleukæmia," *Archiv*, vol. lxxx, No. 1, 1906.—Wende, *Amer. Jour. Med. Sci.*, December, 1901.

Pseudoleukæmia cutis.—Joseph, Arning, Wende, and others have recorded analogous lesions in the skin developing in pseudoleukæmia.

Etiology and Pathology.—The skin lesions may precede or follow the development of leukæmia and pseudoleukæmia, and are an expression of the general conditions.

The skin lesions consist of collections of lymphoid tissue in the corium and subcutaneous tissue. Pinkus believes the pathogenesis of the skin lesions to be the same as that of the spleen and lymph glands, and that they represent hyperplasias of foci of lymphoid tissue normally present in the skin. The treatment of these tumors is part of the conditions in which they occur. In view of the remarkable effect of x-rays upon the deeper-seated tumors in leukæmia and pseudoleukæmia, it is probable that they would quickly dissolve the lesions of the skin.

XI.—MYCOSIS FUNGOIDES[1]

SYNONYMS: *Granuloma fungoides, Granuloma sarcomatodes, Inflammatory Fungoid Neoplasm, Ulcerative scrofuloderm, Eczema tuberculatum, Fibroma fungoides, Lymphoderma perniciosa, Sarcomatosis generalis.*

Mycosis fungoides is a disease of the skin, of slowly malignant course, characterized in the beginning by numerous areas of circumscribed, itching dermatitis, and later by the development of fungoid tumors which tend to ulcerate and ultimately produce fatal cachexia. It was first described by Alibert in 1814, who later gave it the name of mycosis fungoides.

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[1] Stelwagon and Hatch, *Jour. Cutan. Dis.*, 1892, p. 1 (histology and bacteriology, and bibliography from 1885).—Ledermann, *Archiv*, vol. xii, p. 683 (bibliography to 1889).—Morrow, *Jour. Cutan. Dis.*, 1897, p. 65.—Hyde and Montgomery, *Jour. Cutan. Dis.*, 1899, p. 253.—Max Wolters, *Biblioth. Med. Abth. D. II Derm. u. Syph.*, 1899 (bibliography).—Galloway and MacLeod, *Brit. Jour. Derm.*, 1900, pp. 153 and 187 (histology).—Hancock, *Jour. Amer. Med. Assn.*, 1904, vol. xlii, p. 705.—Towle, *Boston Med. and Surg. Jour.*, 1904, vol. cli, p. 629.—Brandweiner, *Monatshefte*, November, 1905.

Symptomatology.—The disease may conveniently be divided into three stages:

First, the premycotic stage.
Second, the stage of infiltration.
Third, the fungoid stage.

They represent the gradual evolution of one pathological process. They usually occur in sequence, and every stage may be of very long duration. But occasionally the fungoid stage abruptly ushers in the disease, and ordinarily in the fungoid stage later lesions typical of the other stages are present.

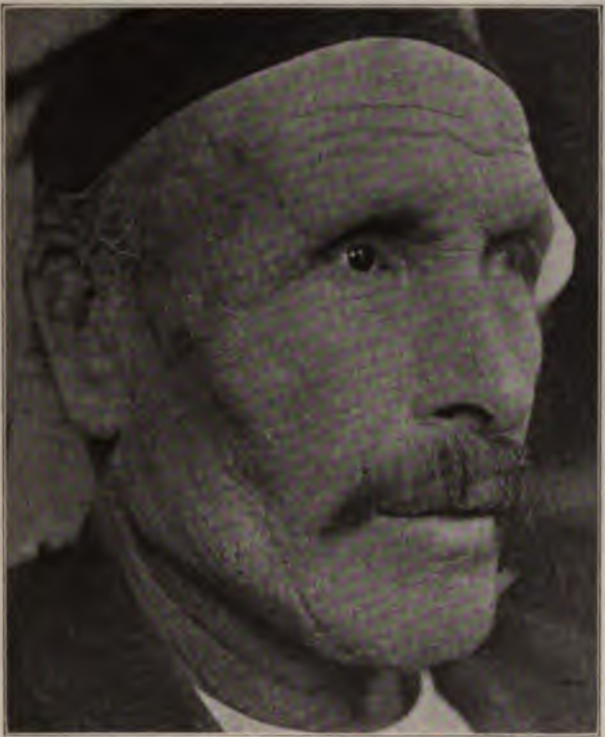


FIG. 320.—MYCOSIS FUNGOIDES, SHOWING DIFFUSE INFILTRATION OF FACE, PREMYCOTIC STAGE. (Author's collection.)

Premycotic Stage.—In the premycotic stage the characteristic feature of the disease is an eruption of circumscribed areas of dermatitis accompanied by intense itching. The form of this inflammation is not fixed. The lesions are usually areas of dry dermatitis which may resemble dry eczema, lichen pilaris, psoriasis, or be simply erythematous or urticarial patches. They may also occur as patches of weeping dermatitis. The patches are ill defined, frequently of circinate outline, and of varying size. The color varies through all the shades of red. Their course is very capricious. They may persist, or they may come and go without regularity.

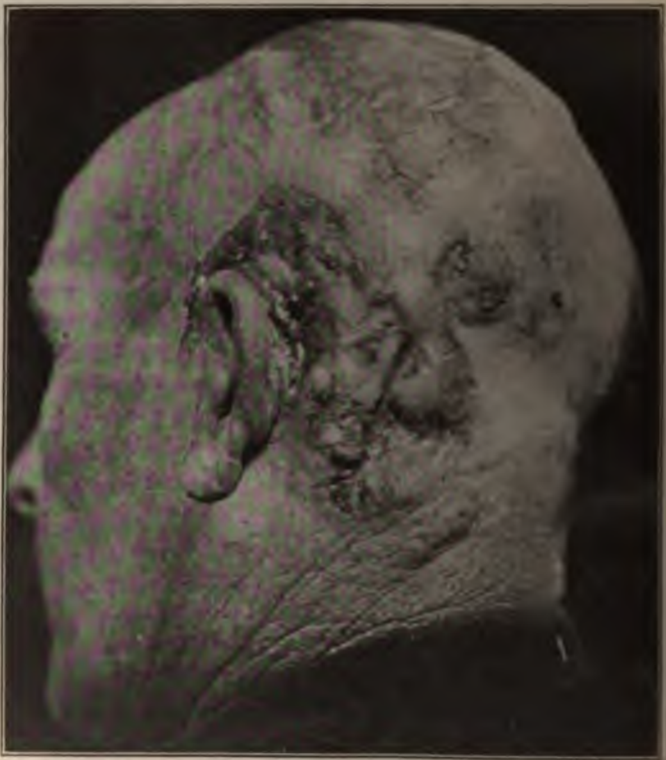
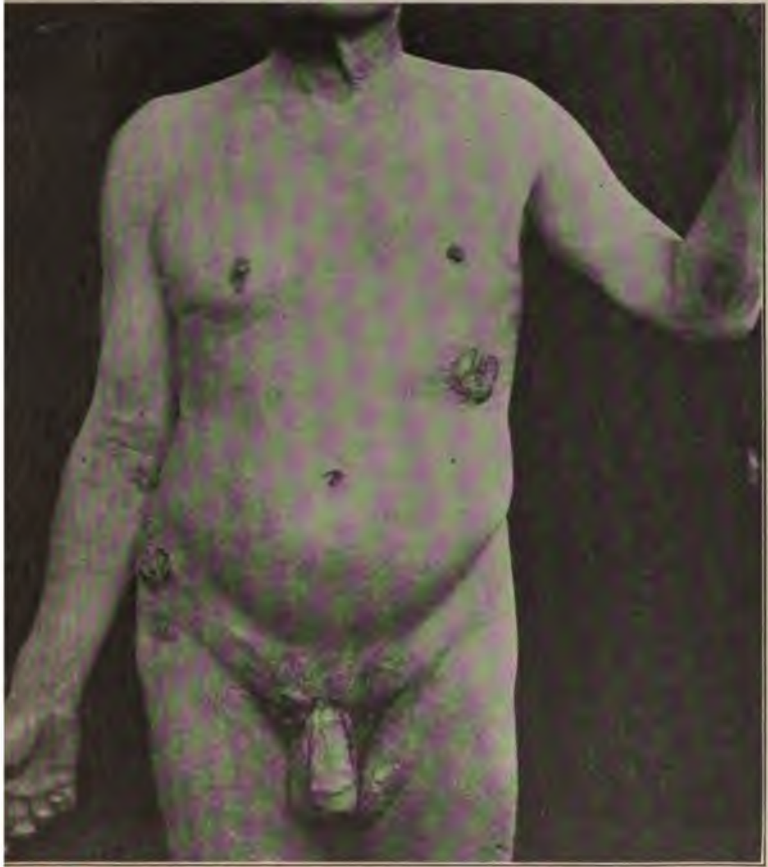


FIG. 320a.—SAME CASE, SHOWING TUMORS OF SCALP, FUNGOID STAGE. (Author's collection.)

Stage of Infiltration.—The duration of the premycotic stage may be short or it may extend over many years. Sooner or later, however, the patches increase in infiltration and there develop sharply defined, elevated plates and nodules either in the previous lesions or elsewhere. The nodules are pea to hazelnut size, and occurring in groups they form irregular, nodular plateaux. Their color varies from pink to dark red or even brown or violet. These lesions are usually of irregular or circinate outline, and they have a characteristic tendency to peripheral spreading and central involution, with the formation of crescentic or yclic figures. These lesions, like those of the eczematous stage, r and disappear, but the disease as a whole is persistent. This like the first, may be of short duration, or it may continue for years.

ungoid Stage.—The stage of infiltration is followed sooner or later e fungoid stage, which is characterized by the development of promi- and later vegetating tumors. The tumors in the beginning are



G. 321.—Same Case, showing Lesions in Various Stages of Evolution. (Author's collection.)

y exaggerations of the nodules of the stage of infiltration, but they ase in size and form sessile, pedunculated, or lobulated tumors up e size of an orange or larger. They are from pink to bright or dull n color, and at first are covered by normal epidermis. Later ulcera- occurs in the tumors. They continue to grow and develop into fungoid, red, tomatolike masses. Such tumors may coalesce into enormous masses of blood-red, suppurating, unhealthy granulations. They are particularly prone to appear about the face, but they may appear upon any part, including the palms and soles. About the face the disease i its late course produces the most horrible deformity. There is usuall adenopathy, but there is almost no tendency for the development of th tumors in the deeper tissues. The fungoid stage is relatively short, an death occurs within from a few months to two or three years. Late i the course of the disease cachexia becomes marked, and the patient die from pyæmia, pneumonia, or some other intercurrent affection t which they are a ready prey. During the first and second stages o the disease severe itching is a characteristic symptom. This may continue to a less extent during the third stage, but it is usually notabl lessened.

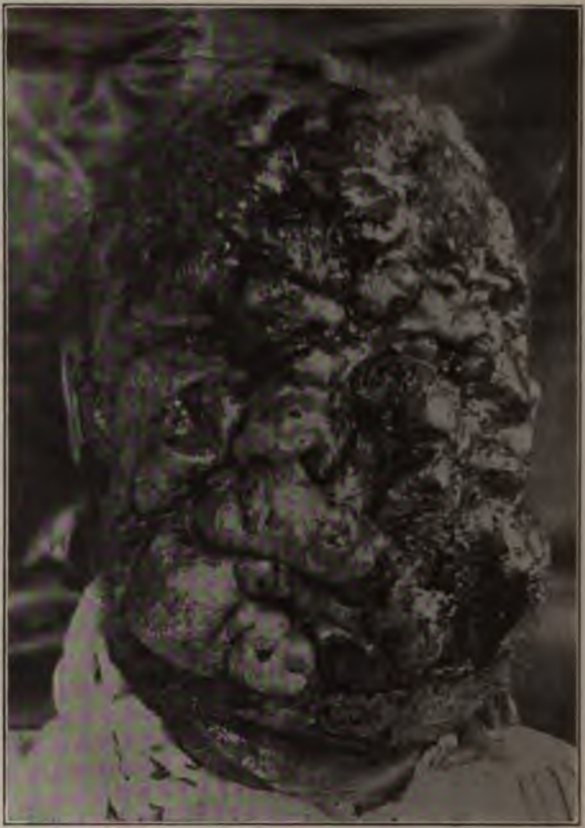


Fig. 322.—Mycosis Fungoides, Late Fungoid Stage. (Breakey's collection.)

iology.—The disease is three times as frequent in men as in
n. Half the cases begin after forty, and few before thirty. Its
ter is suggestive of an infectious disease, but the conveyance of the
e has never occurred, and inoculation experiments have uniformly
egative. There has not been in any of the cases any evidence of
or hereditary predisposition. Some authorities still class mycosis
ides among the sarcomata, but a greater number believe it to be a
loma due to some form of microörganism as yet undiscovered.
logically the disease
e studied in two stages:

) **Premycotic Stage.**—
ogically the earliest
e is a congestion of
pillaries of the corium,
d which there occurs
filtration with various
of cells, the most
ant being similar to
lls of a small round-
sarcoma; plasma and
cells are also present
uch smaller number.
multiform cellular in-
ion is supported by a
te fibrous reticulum. At
tage the epidermis may
rmal, but more often it
various changes such
lema, acanthosis, and
ratosis.

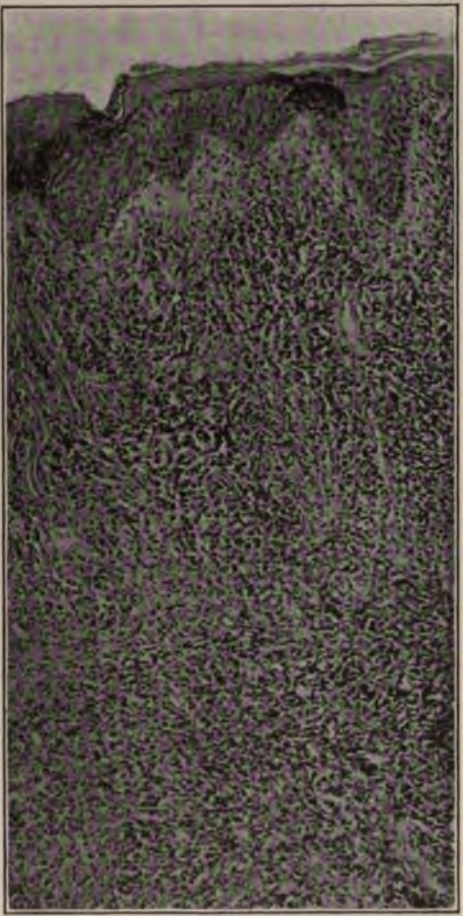


FIG. 323.—MYCOSIS FUNGOIDES.
(C. J. White's collection.)

) **Mycotic Stage.**—The
lomatous tissue gradu-
increases, involves the
e, and finally causes
rowth to project above
rrounding surface; the
mis, which now com-
s to show the effects
retching and impaired
ion, finally ulcerates.
g this stage Galloway and MacLeod have described a crenation
gmentary degeneration of the granulomatous cells accompanied
basophilic degeneration of the collagen and elastin. In one of

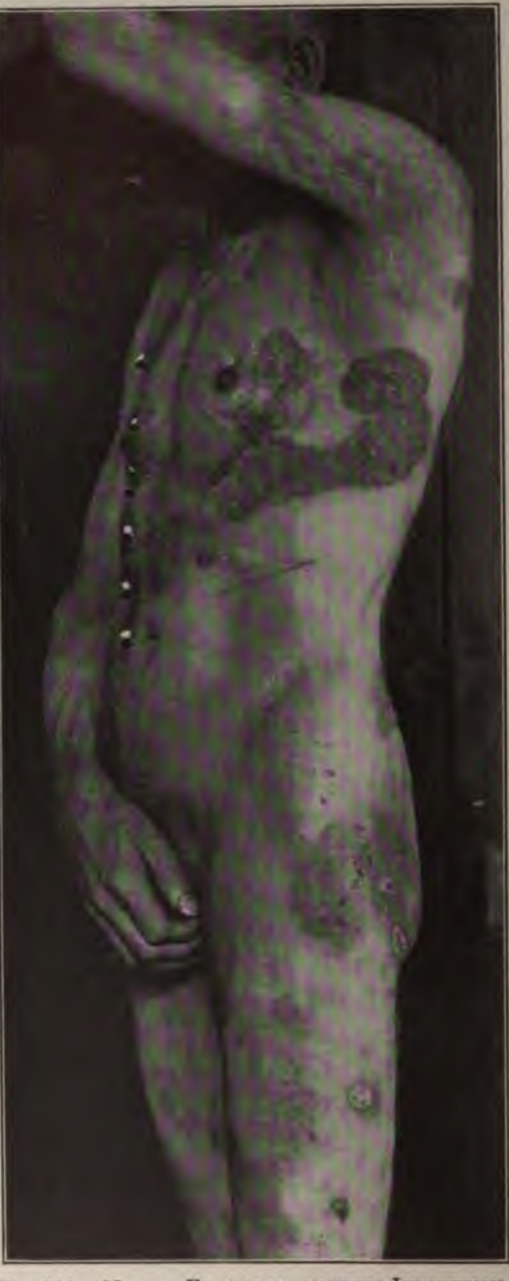


FIG. 324.—MYCOSIS FUNGOIDES, SHOWING INVOLUTION OF FUNGOID LESIONS UNDER X-RAYS. (Author's collection.)

my cases in which mo
of the tumors had disa
peared from x-rays the
was an eosinophilia
high at times as thir
two per cent. Hodar
in two cases found
leucocytosis in the
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which he regards as ch
acteristic.

Diagnosis.—In
eczematous stage a d
nosis cannot be made,
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goid stage the chara
istic infiltrations and
history render the d
nosis easy. The co
of the disease as a w
is unique.

**Prognosis and Tr
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in from a few mo
to fifteen years or m
After the developmen
the fungoid stage, the
tient's expectancy is
duced to a period v
ing from a few mo
to one or two years.

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[1] Hodara, *Monatshefte*, 1904, vol. xxxviii, p. 490.

Treatment has hitherto been practically unavailing in staying the course of the disease, although in one case recovery has been observed after accidental erysipelas (Bazin), and two recoveries have been recorded from the administration of arsenic (Köbner and Geber). Recently reports from all over the world, beginning with a case reported by Jamieson, have announced the symptomatic cure of the disease by the use of x-rays. The skin lesions can be made to disappear entirely, leaving only pigmented areas, and the improvement in many cases has persisted up to the present time, although there is some tendency to relapse unless the treatment is thoroughly carried out. In one case recorded by C. J. White,[1] in which the skin lesions yielded rapidly to x-ray exposures, the patient suddenly developed an acute febrile disturbance and died in a few days. There was found *post mortem* a general septic infection, but White is inclined to attribute the death to acute intoxication resulting from rapid disintegration of the tumors. The exposure to x-rays is not only effective against the tumors, but notably so against the itching.

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[1] White, *Trans. Amer. Derm. Assn.*, 1906.

SECTION XIV

DISEASES OF THE APPENDAGES OF THE SKIN

IN the class of diseases of the appendages of the skin are taken up certain functional and organic affections of these appendages, which are most conveniently grouped according to the structure which is involved. The classification is one of convenience. There are included in it disturbances of entirely different pathological characters, while on the other hand many diseases involving these structures are considered elsewhere.

A. DISEASES OF THE SWEAT GLANDS[1]

I.—HYPERIDROSIS

SYNONYMS: *Idrosis, Ephidrosis, Sudatoria, Polyidrosis.*

Hyperidrosis is excessive sweating independent of the usual physiological causes.

It may be symptomatic or idiopathic, generalized or circumscribed.

Symptomatology.—Symptomatic hyperidrosis occurs in various diseases, like acute rheumatism and pulmonary tuberculosis, and is a part of those diseases. The forms of hyperidrosis which we have to consider are so-called idiopathic universal hyperidrosis and idiopathic circumscribed hyperidrosis.

Universal hyperidrosis is very variable in degree. Ordinarily the attacks come and go; occasionally it is continuous, and it may be so excessive as to cause great prostration. Myrtle[2] and Richardson[3] have recorded cases in which excessive generalized hyperidrosis continued until it produced fatal exhaustion. In Richardson's case it was confined to one side of the body, and was accompanied by erythema of the same side. Universal hyperidrosis is sometimes the result of nutritional disturbances, like obesity, and at other times of nervous disturbances. It is not infrequently seen in very nervous individuals. In rare cases it occurs as the result of a central nervous lesion, or of some obscure trophic disturbance.

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[1] Bouveret, *Thèse de Paris*, 1880 (review and literature).—Pooley, *Ohio Med. Recorder*, vol. v, 1880–81 (review and literature).

[2] Myrtle, *Medical Press*, 1885.

[3] Richardson, *Asclepiad*, 1885, p. 191.

Localized hyperidrosis is usually symmetrical and confined to special areas, the most frequent of which are the palms and soles (either together or separately), the axillæ, and, less frequently, the genitals. Hyperidrosis of these various parts may be moderate or it may be extreme; on the palms and soles, for example, it may be so abundant as to keep the parts continually wet with perspiration, which at times will drip from the fingers. The palms and soles are usually cold, and in the marked cases are erythematous and sodden, and there is often increase of the horny epidermis. On the feet symmetrical keratoses frequently accompany the condition and, from the pain which they cause in walking, render the affection a serious one. The condition may be permanent or it may be temporary.

Unilateral localized hyperidrosis occasionally occurs, and is nearly always confined to the distribution of a nerve. It is seen most frequently in the area of the fifth nerve, and sometimes in the distribution of one of the nerves of the extremities. It is usually accompanied by a faint erythema of the involved area.

Etiology.—Hyperidrosis is produced by disturbances of innervation of the sweat-controlling apparatus. On the hands and feet it is frequently associated with passive congestion of the parts. Most of the patients are of neurotic tendency, and in many there is added to this a lowered condition of the general health from various causes. The condition may be the result of central nerve lesions. Sometimes the condition is congenital. Localized hyperidrosis is at times apparently vicarious, as in conditions where general sweating does not occur, like ichthyosis. The localized cases occurring in the distribution of cutaneous nerves are usually associated with neuritis or some other lesion of the nerve.

Treatment.—In the generalized forms treatment is directed to the relief of the underlying systemic disturbances. In the idiopathic cases it is, as a rule, only partially successful. In the localized forms, underlying conditions require treatment as far as possible, but most benefit is to be given by local applications. Local applications which may be used with benefit are tannic acid, alum, or zinc sulphate, from 1 dram to 1 ounce to a pint of water, or, if the parts are not excoriated, the same substances in equal parts alcohol and water. Another useful application is one-per-cent solution of formalin in water. The parts should be washed, and then bathed in one of these solutions for several minutes twice daily, and afterwards powdered with an antiseptic dusting powder, of which powder boric acid, either with or without one- or two-per-cent salicylic acid, is best.

A favorite method of treating hyperidrosis of the feet is by the use of diachylon ointment, or an ointment of tannic acid, one or two drams to the ounce of simple ointment. The parts are first washed in soap and water, and then rubbed dry. The ointment is spread thickly on cloths, and these are applied to the surfaces. After twelve hours the ointment is wiped off with a towel, the surfaces dusted with boric-acid powder, and a new dressing applied. This is continued until the epidermis freely exfoliates—for from ten days to two weeks—after which the parts should be cared for daily with an astringent lotion and a dusting powder.

None of these methods does more than give temporary relief, and all are very inferior to the application of x-rays. The functional activity of the sweat glands, as of the sebaceous glands, can be inhibited to as great a degree as is desired by cautious exposures to x-rays. In the treatment of hyperidrosis with x-rays, the parts should be exposed cautiously from one to three times a week until the excessive sweating ceases. It is not necessary to produce an erythema. If, after improvement, recurrence begins, the exposures are to be repeated. I have used the method in many cases. In some of them the results have remained permanent for a year or more, in other cases the patients have returned occasionally for a few exposures, but practically in all cases the results meet the exacting expectations of the patients.

II.—ANIDROSIS

Anidrosis is diminution in the secretion of sweat. It is a relative condition, and may exist in all degrees up to complete cessation. In its extreme degrees it is a rare condition.

Symptomatology.—It may be localized or general. It is often seen in association with diseases where there is an increased secretion of urine, and not infrequently it is to a certain extent seen in conditions of defective innervation or of malnutrition. The localized form occurs in association with skin diseases in which sweating ceases in the affected areas, as in anæsthetic leprosy, general and circumscribed scleroderma, psoriasis, and in some cases of eczema and pityriasis rubra pilaris. At other times localized anidrosis follows nervous lesions, either peripheral or central, and occurs in the area of the affected nerves. General anidrosis is a characteristic symptom of ichthyosis, where it is due to congenital failure of development of the sweat glands. In a case described by Tändlau, general anidrosis, due to congenital defect in the sweat glands, was associated with defective development of the hair and teeth, and other developmental defects.

Permanent failure in the sweat secretion, as in xeroderma, produces a harsh, dry skin which is vulnerable to external irritants, and is apt to be subject to seasonal pruritus and to dermatitis.

Treatment.—In the symptomatic forms of anidrosis treatment is directed to the underlying disturbances. In temporary anidrosis, as in generalized eczema, often great relief in the itching from which these cases particularly suffer can be obtained from the administration of pilocarpin. In the permanent forms of anidrosis nothing can be done to overcome the condition, but the unfavorable effects upon the skin due to lack of lubrication can be largely overcome by the use of emollient baths or inunction of oils or fats.

III.—BROMIDROSIS[1]

Bromidrosis is sweat of offensive odor. It may be due to primary alterations in the composition of the sweat or to decomposition after excretion.

In nearly but not all cases it is associated with hyperidrosis, and is closely bound up with that condition.

Symptomatology.—It may be general or local. The general forms are most frequently symptomatic of diseases of metabolism, like acute rheumatism, scurvy, or uræmia. *Uridrosis* is applied to sweating of urinous odor. It is found chiefly in uræmia, but also occurs in cholera, and occasionally in other diseases. It is due to vicarious excretion by the skin of the urinary constituents, especially urea and the urinary chlorides. The amount of urea excreted by the skin is sometimes so great as to be deposited upon the surface in white crystals. Rarely bromidrosis results from ingesta. Substances of penetrating odor which are excreted by the perspiration may produce temporary bromidrosis. Among these are asafœtida, sulphur, benzoic acid, onions, garlic, and musk. There are also some personal variations in the odor of the sweat, and occasionally this is of sufficient degree to produce slight bromidrosis. Racial variations in the odor are well known, as especially illustrated by the peculiarity in the odor of the sweat of the negro and Mongolian races. In rare cases, especially in association with nervous diseases, abnormal but agreeable odor of the sweat has been observed. Hammond called attention to cases of emotional disturbance in which the sweat had the odor of violets or pineapple.

Localized forms of bromidrosis are not uncommon. The areas involved are the palms and soles, axillæ, and, less frequently, the genitocrural region. These may be involved together or independently of each other. Localized forms of bromidrosis are almost invariably an accompaniment of hyperidrosis. Local bromidrosis may be due to unusual composition of the sweat resulting from the same sort of disturbances that produce general bromidrosis, but it is usually the result of the decomposition of sweat by the bacterium fœtidum of Thin. This is a micrococcus, which can readily be demonstrated by drying the sweat from the feet upon a slide and staining with methylene blue, and, according to Moore, is a soil bacterium which reduces nitrates, sulphates, and phosphates into their corresponding nitrites, sulphites, and phosphites.

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[1] Monin, *Jour. Cutan. Dis.*, 1885, p. 211, *Abs.*

Bromidrosis, except upon the feet, rarely amounts to more than slight annoyance. Bromidrosis of the feet, however, in extreme cases is so offensive as to exclude the victims from society.

Treatment.—The treatment of bromidrosis upon other parts than the feet is the same as that of hyperidrosis. In severe cases affecting the feet it is treated by various antiseptic applications. Bathing the feet in alcohol of varying strengths, in bichloride solution from 1:1,000 to 1:5,000, in solution of formalin or of potassium permanganate from 1:100 to 1:500, are among the best applications. This should be done twice daily, and should be followed by thorough dusting of the feet with boric-acid powder. At the same time care should be given to the shoes and stockings. The stockings should be dredged in boric acid, should be changed daily, and the shoes likewise should be powdered. They should be large and loose, to allow for some circulation of air, and soon after beginning treatment it is well to have the patient begin the wearing of a new pair of shoes. As in hyperidrosis, x-rays are especially efficient in controlling the sweating.

IV.—CHROMIDROSIS[1]

Chromidrosis is the term applied to the excretion of pigment by the sweat. The pigment probably in many cases is excreted by the sebaceous glands as well as the sweat glands, and the two conditions cannot be dissociated.

Symptomatology.—Chromidrosis occurs as localized forms of colored sweating. It appears upon the eyelids, especially the lower, the nose, and other parts of the face, and occasionally on the neck, back, breasts, axillæ, hands, and genitocrural regions. The area around the eyes is most frequently affected. In a very few cases the affection has been localized to sharply circumscribed areas of the hands and other parts of the extremities. The discoloration is usually black or brown, but may vary through shades of blue, and very rarely cases of red, green, and yellow sweating have been observed. There is in these cases a true excretion of pigment in the sweat and sebum which collects upon the skin in a granular deposit. The pigment is readily dissolved by ether or alcohol, but is very slightly soluble in water. The discoloration produced by the deposit sometimes appears suddenly, but as a rule gradually develops upon the surface. The condition may be associated with peculiar pigment in other excretions, including vomitus, fæces, urine, and milk. *Green sweat*, and occasionally bluish sweat, may result from the ingestion of copper or from its absorption from the fumes; it is seen chiefly in copper workers. Its character can be positively established only by chemical determination of copper in the pigment excreted in the sweat.

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[1] Foot, *Dublin Quarterly Jour. Med. Sci.*, August, 1869, December, 1873.—Heidingsfeld, *Jour. Amer. Med. Assn.*, December 13, 1902 (review and bibliography).

Etiology.—Most of the cases of supposed colored sweating are due to the formation of bacterial pigments in the sweat after excretion—pseudochromidrosis (*q. v.*)—or to malingering. It is well established, however, that pigments may be excreted in the sweat. True chromidrosis nearly invariably occurs in hysterical or highly neurotic individuals, and nearly always in association with chronic constipation and indicanuria. The great majority of the patients are women, the ages ranging from fifteen to fifty, most frequently young adults. The theory that the pigment is indigo, produced by the oxidation of indican after its excretion, is disproved, at least in some cases, by the failure to obtain indigo reaction from the pigment.

Diagnosis.—The only condition with which chromidrosis can be confused is pseudochromidrosis, from which it can be distinguished by careful observation of the method of formation of the pigment.

Prognosis and Treatment.—The affection gets well, but it may recur. Occasionally it persists in recurrent attacks for several years.

Treatment is directed to the relief of the underlying causes, of which constipation is the most important. Local treatment has no effect, but the discoloration can be removed by washing with chloroform or alcohol.

V.—BLOODY SWEAT OR HÆMATIDROSIS[1]

Hæmatidrosis is excretion of blood or blood pigment through the sweat glands. It is, as a rule, one of the manifestations of purpura, and is analogous to the hæmorrhages that occur from purpura in other structures. It is sometimes a manifestation of highly emotional disturbances, and is associated at times with the bleeding stigmata of hysterical subjects.

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[1] Hyde, "Bleeding Stigmata," *Jour. Cutan. Dis.*, 1897, p. 557 (review and bibliography).

VI.—PHOSPHORESCENT SWEATING OR PHOSPHORIDROSIS

Phosphorescent sweating is sometimes observed in advanced tuberculosis or cancer. It may occur in individuals who are taking phosphorus, or who have eaten putrid fish in which phosphorescent products have been formed by photogenic bacteria. The phosphorescence in the sweat may be associated with the same phenomenon in the urine and other secretions.

VII.—PSEUDOCHROMIDROSIS

This is discoloration in sweating areas from the formation of pigment by bacteria growing in these areas. It is not uncommon in the axillæ, where it is associated with lepothrix. It occurs as a formation of orange-yellow to red granules and concretions upon the hairs, and is produced probably by various pigment-forming bacteria, including the bacillus prodigiosus. The pigment can readily be removed by alcohol or ether.

VIII.—MILIARIA[1]

Miliaria is a sweat eruption produced by the retention of sweat in the epidermis at the mouth of the sweat follicles. It occurs in two forms, miliaria crystallina and miliaria rubra. The first is characterized by noninflammatory lesions, the second by inflammatory lesions. The two affections may be compared to comedo and acne, m. crystallina being a retention lesion in the sweat follicles and m. rubra a retention lesion plus inflammation of the tissues around the follicle.

Miliaria Crystallina (SYNONYMS: *Sudamen, Sudamina*).—Miliaria crystallina is characterized by an abundant eruption of closely set but discrete, minute, acuminate, noninflammatory vesicles which are filled with sweat. They are translucent, glistening, and show by their appearance the watery character of their contents. The vesicle walls are thin and are easily broken, but do not rupture spontaneously. They disappear by absorption in a very few days, and are followed by slight desquamation. The affection usually lasts from a few days to a week, but may be continued by the appearance of new crops of vesicles. The eruption may occur upon any part of the body, but it is most frequent on the trunk, especially upon the anterior surface. It is without subjective symptoms. Miliaria crystallina may be produced by excessive sweating or by a sudden onset of sweating after a period in which sweating has been in abeyance. It occurs near the termination of acute febrile disturbances, like typhoid, malaria, septicæmia, and acute rheumatism, in which critical sweating occurs, or in the course of depressing disease, like tuberculosis, in which there is free sweating. The affection may be produced by clogging of the sweat orifices by horny epithelium or by swelling of the sodden horny epidermis around the sweat follicles, and the consequent occlusion of the orifices.

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[1] Robinson, *Jour. Cutan. Dis.*, 1884, p. 362.—Török, *Monats.*, 1891, vol. viii, p. 437.—Pollitzer, *Jour. Cutan. Dis.*, 1893, p. 50.

Miliaria Rubra (SYNONYMS: *Prickly Heat, Heat Rash, Red Gum, Lichen tropicus, Strophulus*).—Miliara rubra is an eruption of inflammatory miliary vesicles and papules at the mouths of the sweat follicles. It differs from miliaria crystallina by the addition of the inflammatory feature. It is familiar as prickly heat.

The typical lesions of miliaria rubra are superficial, thin-walled, small, acuminate vesicles upon a slightly inflamed base—*miliaria vesiculosa.* The contents of the vesicles at first are transparent, and later become opalescent. Like the lesions of miliaria crystallina, the vesicles do not tend to rupture spontaneously, but dry up quickly and are followed by slight desquamation. Along with the vesicular lesions in miliaria rubra there are usually small papular or papulo-vesicular inflammatory lesions. This type of lesion may dominate the eruption—*miliaria papulosa*—but ordinarily the two types of lesions are both present, with the vesicular type most prominent. The eruption of miliaria rubra occurs as an abundant crop of closely set but discrete lesions, frequently giving the involved area a diffuse, pinkish-red color. The eruption occurs by preference upon the trunk, especially upon the back, but it may occur anywhere. The affection tends to disappear in the course of a week or ten days, but may be continued by the eruption of fresh crops of lesions. In miliaria rubra there are burning, prickling, or itching sensations which may be of considerable intensity. Occasionally, from infection, the lesions of miliaria rubra become purulent, and it is not infrequent for small abscesses to form in the sweat follicles. It is not uncommon, also, for a dermatitis, produced by the irritation from the sweat, to accompany miliaria rubra. This is most likely to develop in the form of intertrigo in the flexures.

Etiology and Pathology.—Both miliaria crystallina and miliaria rubra are sweat eruptions, the first occurring in the course of prostrating diseases accompanied by severe sweating, the second in association with exposure to high temperature. Miliaria rubra is usually seen in summer, and is most frequent in children or in adults who sweat freely. The lesions of miliaria crystallina are produced by the retention of sweat under the horny epidermis at the mouths of the sweat follicles. This may be produced by the growing of the horny epidermis over the openings of the sweat follicle, or by the swelling of the horny cells and the occlusion of the orifices of the sweat follicles. To my mind there seems every reason to believe that the primary process in miliaria rubra is the same, and that the inflammatory symptoms are secondary to the irritation produced from the retained sweat. The histological structure of the lesions, as a rule, indicates this pathology. It must be said, however, that this simple pathology of miliaria rubra does not express the universal opinion. Robinson holds that the disease is an affection of the epidermis, and not of the sweat glands alone, although stating that there is always excessive functional activity in the sweat glands and that the lesions occur around the sweat glands, while Török holds that the disease is entirely independent of the sweat glands. All of the larger facts in the disease make it impossible, to my mind, to accept these explanations, or to escape the obvious explanation of the condition.

Diagnosis.—The association of both forms of miliaria with excessive sweating will always give the clew to the diagnosis. The existence of glistening, very superficial vesicles, and the evanescent course and trivial character of the eruption, are sufficient to distinguish it from other vesicular dermatoses.

Treatment.—Miliaria crystallina requires no treatment. Bathing with dilute alcohol, followed by powdering the surfaces with boric-acid or other bland dusting powders, hastens the disappearance of the condition.

In miliaria rubra the itching is stopped, and the resolution of the affection is hastened by bathing in dilute alcohol, followed by the use of a dusting powder. Calamine lotion dabbed over the surface after bathing with dilute alcohol is another excellent application. In those who are subject to miliaria its occurrence can be prevented by frequent bathing, followed by an alcohol rub and the application of a bland dusting powder.

IX.—MILIARY FEVER OR SWEATING SICKNESS[1]

This term is applied to a constitutional disease characterized by severe sweating and the occurrence of miliaria. Severe epidemics of it occurred in England in the fifteenth and sixteenth centuries, and the last epidemic in France in 1887. It is an infectious disease, with high fever, great prostration, and often a fatal ending. Its character is undetermined.

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[1] Nothnagel's Handbook.—Vignol, *Lemaine Médicale*, xxvii., No. 2, pp. 13-24.

X.—POMPHOLYX

SYNONYMS: *Cheiropompholyx, Dysidrosis.*

Pompholyx is an affection occurring upon the hands and feet, characterized by deep-seated, clear, grouped vesicles which appear suddenly, have narrow red areolae, and run a short course.

Symptomatology.—The disease begins by the rapid development of groups of deep-seated vesicles upon the palms and between the fingers, and less frequently upon the same parts of the feet. In some cases the backs of the hands may also be covered by the lesions. The eruption is symmetrical. The vesicles are at first transparent, gradually become opaque, and may or may not become pustular. The lesions are covered by a thick layer of horny epidermis, and they may be without elevation. Usually they are round, and sometimes they form prominent bullæ. Grouped lesions tend to coalesce. The lesions disappear by absorption in the course of one or two weeks; the epidermis exfoliates, and there is left a reddened base which slowly disappears. The lesions may be few or numerous; when very numerous they may cause diffuse swelling of the hands. The affection is characterized by pricking, burning, and itching, usually slight but occasionally severe, and in the extensive cases the hands are stiff and tender. The eruption disappears spontaneously in from ten days to two weeks, but may be continued by recurrences. The disease is liable to recur in individuals who are subject to it.

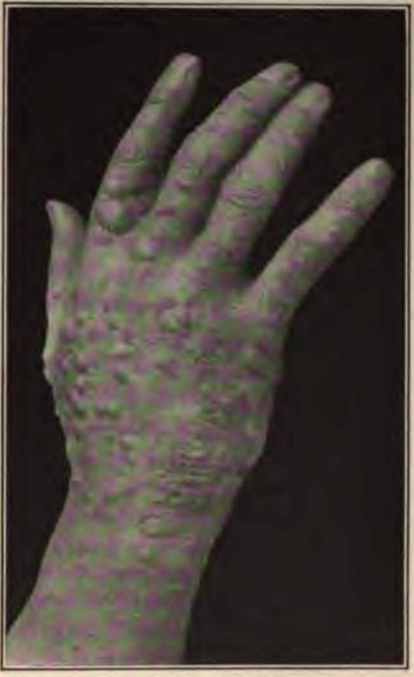

FIG. 325.—CHEIROPOMPHOLYX. Location on back of the hand unusual. (C. J. White's collection.)

Etiology[1] **and Pathology.**—Pompholyx usually occurs in patients who have hyperidrosis of the palms and soles. It is rare in childhood and old age, and more frequent in women than in men. It is most frequent in warm climates, and in individuals who are enervated. It is rather uncommon in cooler climates.

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[1] Engman, *Med. Review*, November 11, 1899.

Anatomically, the lesions consist of spaces in the epidermis filled with clear contents in which leucocytes subsequently appear. The inflammatory changes in the corium are very slight. A connection between the lesions and the sweat follicles can frequently be demonstrated. Tilbury Fox, one of the original describers of the disease, regarded it as a sweat eruption, and in my opinion that view is correct, the affection being exactly analogous to miliaria upon other parts, but presenting clinical differences on the palms and soles because of the thickened layer of epidermis under which the sweat is retained. This is contrary to the general opinion, which regards pompholyx as a disturbance of neurotic origin and places it in the class with herpes, pemphigus, and the other angioneurotic eruptions.

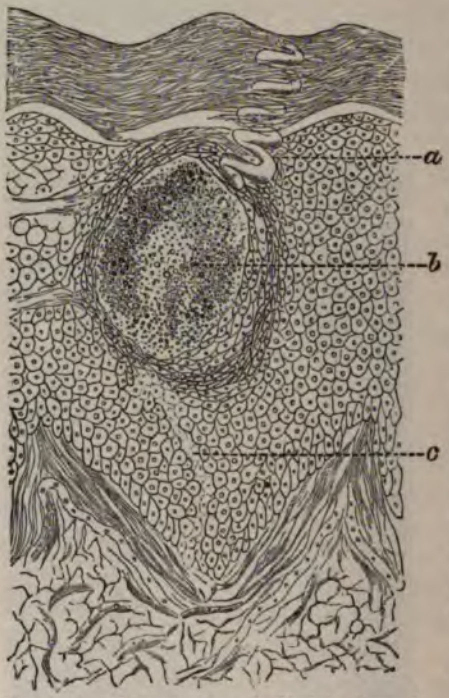

FIG. 326.—POMPHOLYX. *b*, Vesicle formed in the interpapillary portion of rete directly in the course of the sweat channel *a* and *c*. (Crocker.)

Diagnosis.—The appearance of groups of deeply embedded, transparent vesicles upon the palms, or upon the palms and soles, accompanied by relatively slight inflammatory reaction and little subjective disturbance, and running a short course of ten days or two weeks, are characteristic features which distinguish the affection from eczema or impetigo, with which it might be confused.

Treatment.—The local treatment is similar to that for hyperidrosis pedum. Calamine lotion or one-per-cent solution aluminum acetate is useful as an astringent and to relieve the subjective sensation. Relapses and recurrences may be prevented by bathing the parts in astringents, such as are used for hyperidrosis, and by the administration of tonics and the use of other measures to build up the patients. Iron, quinine, strychnine, and especially arsenic are useful tonics in the affections.

XI.—HIDROCYSTOMA[1]

Hidrocystoma is an affection characterized by an eruption of deep-seated vesicles which are cysts of the sweat ducts. The disease was first described by Robinson in 1884, and many studies of the disease have been made since, although it is not common.

Symptomatology.—The lesions are tense, shining, obtuse, deep-seated, thin-walled vesicles from the size of a pinhead to a pea. The smaller lesions, like those of pompholyx, resemble boiled sago grains embedded in the skin, but the lesions are not inflammatory like those of pompholyx, and the contents remain clear and dry up without becoming

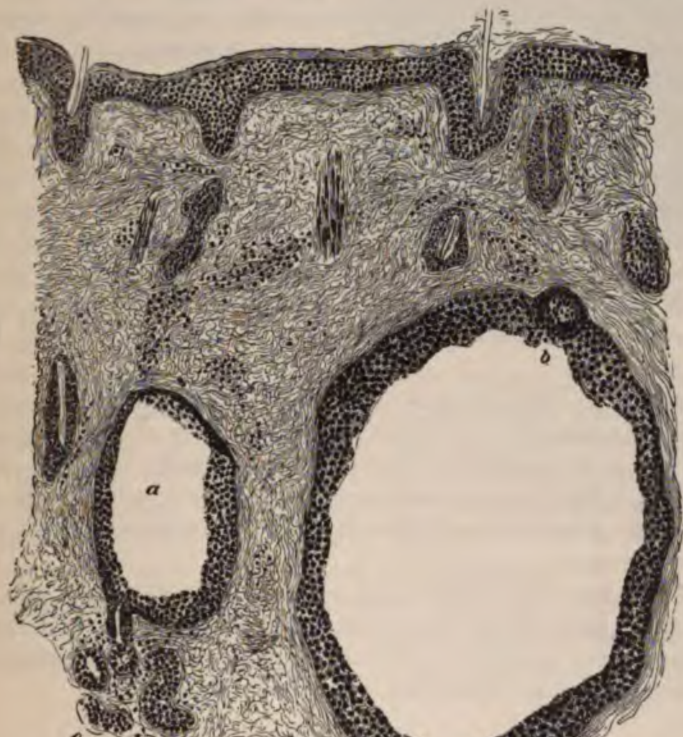

FIG. 327.—*a*, Large and small cyst; *b*, excretory sweat duct at place of obstruction; *c*, coil of sweat gland. (Robinson.)

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[1] Robinson, *Trans. Amer. Derm. Assn.*, 1884.—Morrow's System, vol. iii, p. 780.—*Jour. Cutan. Dis.*, 1893, p. 293.—Jackson, *Jour. Cutan. Dis.*, 1886, p. 1.—Bassàget, *Thèse de Paris*, 1896 (histology and complete review).

yellow. The eruption occurs on the face, especially on the nose, cheeks, lips, chin, around the eyes, and on the lower part of the forehead. Robinson has not seen it upon other parts than the face. Upon the affected parts the lesions are usually discrete but closely set, and they may be very abundant. There may be very slight sensation of tension or prickling. An attack of the disease lasts one or two weeks and then disappears by absorption of the contents of the vesicles, leaving the surface normal or slightly pigmented, but the disease may be prolonged by relapses, and tends to recurrence.

Etiology and Pathology.—The affection occurs in those who perspire very freely and who, like washerwomen, are especially exposed to warm, moist atmosphere. It is usually seen in summer. It occurs most frequently in women of middle or old age who perspire freely. The affection is apparently primarily a sweat eruption, but in some cases there is in Robinson's opinion a nervous element. Anatomically the disease consists of cysts of the sweat ducts filled with sweat. These are primarily situated deep in the corium, but the larger ones closely approach the surface. According to Robinson the cysts are not simply passive dilatations of the ducts, as is shown by the fact that even the smallest ones are always lined by several layers of epithelium. There are few or no inflammatory changes, and the epidermis over the lesions is normal.

Diagnosis.—The affection is to be differentiated from pompholyx and adenoma sebaceum. Pompholyx does not occur on the nose, and the lesions are in the epidermis. The lesions of adenoma sebaceum are persistent, while the lesions of hidrocystoma disappear in the course of two weeks. The histological pictures in the two conditions are, of course, entirely different.

Treatment.—The lesions upon puncture are easily evacuated. After puncture the parts should be sponged twice daily with an astringent solution, like alcohol containing two-per-cent resorcin, and then dusted with an astringent powder, like equal parts bismuth and talcum.

XII.—GRANULOSIS RUBRA NASI[1]

Granulosis rubra nasi is a clinical entity described and named by Jadassohn in 1901 from a study of seven cases, although a case had previously been described in 1900 by Luithlen.

Symptomatology.—The eruption occurs typically on the nose in the form of an ill-defined, reddish patch, dotted with minute macules and maculo-papules. The surface is usually glistening and moist. The lesions are hyperæmic, and the color disappears entirely upon pressure. Occasionally the papules become pustular, but there is no scarring. In most cases the eruption is limited to the nose, but it may involve the pper lip, the cheeks, and the eye- rows. Its course is very chronic, eginning in childhood and tend- ng to spontaneously disappear hen adult life is reached. Le- ions of hydrocystoma have oc- urred as a complication of the ndition. The patients have been ildren, and in most cases boys. he one exception is a case of inkus's in a man of fifty-nine.

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[1] Luithlen, *Kaposi's Festschrift*, 1900, p. 709.—Jadassohn, *Archiv*, 1902, vol. lviii, p. 145.—MacLeod, *Brit. Jour. Derm.*, 1903, p. 131.—Ormsby, *Jour. Cutan. Dis.*, 1905, p. 1830.

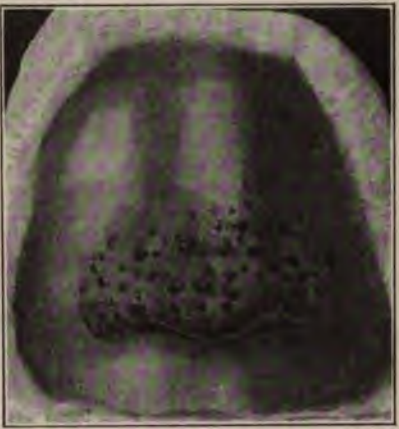

FIG. 328. — GRANULOSIS RUBRA NASI. (Photographic reproduction of Luithlen's plate.)

The disease is distinctly asso- ated with hyperidrosis and poor eripheral circulation. Anatomic- ly the changes are those of an flammatory infiltration around e capillaries of the sweat glands. Definite changes are found in the veat glands; the coils are dilated and contain a finely granular *débris,* nd their walls are greatly thickened. The conditions with which the isease might be confused are lupus erythematosus, lupus vulgaris, osacea, hydrocystoma, and pompholyx, but the affection is sufficiently haracteristic to readily distinguish it from these. It is most likely o be regarded as an atypical form of hydrocystoma.

Treatment.—Treatment has hitherto had little effect upon the ffection. Locally astringents, like lotio alba or preparations of ich- hyol, should be of temporary benefit. X-rays might be of benefit.

B. DISEASES OF THE SEBACEOUS GLANDS

I.—SEBORRHŒA

SYNONYMS: *Steatorrhœa, Stearrhea, Seborrhagia, Fluxus sebaceus, Hyperidrosis oleosa.*

Seborrhœa is hypersecretion of fat by the skin.

It is commonly divided into seborrhœa oleosa and seborrhœa sicca.

Seborrhœa oleosa.—Seborrhœa oleosa consists in abnormal oiliness f the skin. As ordinarily seen, it causes the skin to look greasy and shiny, but occasionally the greasiness is so abundant that the oil will accumulate on the surface in minute droplets. It occurs on the face, especially on the nose and the flush area of the face. In addition to the oiliness, the complexion is more or less muddy and pasty looking; the mouths of the sebaceous glands are patulous, and the texture of the skin coarse; and the skin is with difficulty kept clean on account of the readiness with which dust adheres to it. There is often more or less congestion of the nose, and to a less degree of the other involved areas, and the parts are apt to be cool to the touch. Acne lesions are not infrequently an accompaniment of the condition. Frequently also it is complicated by the presence of slightly reddened areas covered with fine, glossy scales; in such cases we have seborrhœic dermatitis superadded to the simple seborrhœa. Seborrhœa also occurs on the scalp, where it produces increased greasiness of the hair or, if the patient is bald, gives the skin a smooth, shiny, and, it may be, greasy appearance. It may also involve the trunk. This is seen especially in negroes, but may occur in other individuals, regardless of race, who perspire very freely, or whose sebaceous glands are unusually well developed.

Etiology and Pathology.—Seborrhœa oleosa may be regarded as excessive functional activity of the fat-producing glands analogous to hyperidrosis, and the most important factor in its causation is an abundant development of the sebaceous glands. Seborrhœa oleosa occurs most frequently during adolescence with the sebaceous glands are most active, but it may occur at any period of life. In adult life it is commonest in women in whom constipation and uterine disturbances are usually the exciting cause. Indeed, all of the disturbances which cause reflex flushing of the face and produce such affections as acne rosacea may excite seborrhœa.

Seborrhœa oleosa is a functional disturbance chiefly involving the sebaceous glands. Unna has suggested the novel doctrine that the fat of the skin is supplied by the sweat glands, and has suggested the name hyperidrosis oleosa for seborrhœa. Other studies have not borne out his view. There is no doubt, however, that part of the fat of the skin is secreted by the sweat glands, and in seborrhœa oleosa a part, but only a small part, of the oil is doubtless secreted by the sweat glands.

Treatment.—In the treatment of seborrhœa oleosa attention should be given to correcting any constitutional disturbances, but the removal of the seborrhœa depends almost entirely upon local treatment. The applications used are astringents like those used in acne, particularly those containing sulphur and resorcin. The best applications are lotio alba and Vleminckx's solution, which are used in the manner described under acne. Bathing the skin with green soap or in alcohol removes the grease, and this is well followed by a dusting powder containing one part sulphur to four or five of talcum.

Seborrhœa sicca.—Seborrhœa sicca is commonly described as an affection of the sebaceous glands in which there is an alteration in the composition of the sebaceous matter, resulting in its accumulation on the surface in greasy, yellowish scales. Unna and his followers have demonstrated that seborrhœa sicca is an inflammatory disease of the skin, and is a definite clinical entity, of which the condition described as seborrhœa sicca is one of the manifestations; seborrhœa sicca is seborrhœic dermatitis (*q. v.*).

The only condition which the term seborrhœa sicca accurately describes is the accumulations of greasy scales which occur upon the skin in infancy. Throughout early infancy the sebaceous glands are unusually active. This is frequently shown by the accumulation upon the scalp of dirty, greasy crusts. Their accumulation is due to lack of care, and they are readily removed by oil or washing with soap and water. This condition is frequently the starting point for a dermatitis which, if not identical with, is indistinguishable from seborrhœic dermatitis.

II.—ASTEATOSIS

Asteatosis is deficient secretion of fat. It occurs in conditions characterized by dry, harsh skin, like ichthyosis, in which anidrosis also occurs. Anidrosis and asteatosis are so closely related that they cannot be separated, and they are considered as one condition under anidrosis.

III.—MILIUM

SYNONYMS: *Grutum, Strophulus albidus, Acne albida, Tuberculum sebaceum.*

Symptomatology.—Mila are small whitish tumors of the skin formed by the accumulation of inspissated sebum beneath the horny epidermis. They are familiar lesions as the small, white masses which are seen very frequently on the cheeks below the eyes. They occur as firm, white or yellowish papules usually of the size of a pinhead, which are very superficial and whose contents can readily be pressed out after incision. They are found most frequently around the eyes and on the cheeks and forehead, but may occur elsewhere. Frequently they are seen on the scrotum, and they may occur on the penis. After reaching the size of a pinhead, or a little larger, they usually remain stationary. Occasionally they become as large as a small pea. They are ordinarily few, but at times they occur in large numbers. As a rule they remain discrete, but the lesions may coalesce into masses the size of a pea or perhaps larger. These larger lesions are usually yellowish or dirty yellow, and they may become quite hard from the deposit of calcareous salts, chiefly calcium phosphate and carbonate, forming the so-called cutaneous calculi.

Etiology and Pathology.—Milia are common in young adults, especially in association with comedones. They are not infrequent in nursing infants upon the face, and occasionally they are present at birth. They are sometimes produced as result of other lesions of the skin, as at the site of pemphigus bullæ, in scars, and after inflammatory processes like erysipelas. They are a constant accompaniment of lymphangioma tuberosum multiplex.

A milium is most frequently the result of the growth of the horny epidermis over the mouth of the sebaceous follicle, which is followed by the retention of the sebaceous matter beneath the horny epidermis. These lesions are thus only slightly different from comedones. According to Robinson there is another form of milium which is the result of misplaced embryonic epithelial remnants from the hair follicles or from the rete which by their growth produce lesions consisting of masses of imperfectly cornified cells. Milium can only be confused with xanthoma. The lesions are white or yellowish rather than yellow, and are readily evacuated upon incision.

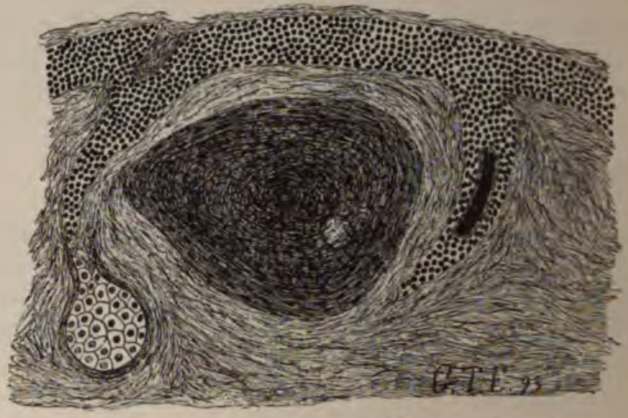

FIG. 329.—SECTION THROUGH A MILIUM. (Elliot.)

Treatment.—After incision the lesions are cleaned out by a little pressure, and the sac may then be touched with carbolic acid or tincture of iodine to prevent recurrence. Where they occur on the face of infants, or on the scrotum, they are readily removed by the use of soap and water.

IV.—CONGENITAL MILIUM IN PLAQUES[1]

Under this term Crocker has described two cases of his which corre-nd histologically to the embryonic type of milium of Robinson. Other es have been reported under different names by Hans Hebra, Wilson, l Colcott Fox.

The condition consists of plaques of reddish-yellow color, which are rply circumscribed, of irregular outline, and slightly elevated, and e a shiny, granulated surface composed of aggregations of yellowish, point-sized papules. The patches occur on the head and face, those the scalp being quite hairless. They are present at birth, and remain tionary. Microscopically the papules consist of masses of nucleated thelial cells inclosed in a fibrous capsule and situated superficially the corium, the microscopical structure leading Crocker to class the ection with the milium due to the misplaced epithelium which Robin- has described.

V.—SEBACEOUS CYST

Synonyms: *Atheroma, Steatoma, Wen.*

Symptomatology.—Sebaceous cysts occur as rounded or oval, rply defined tumors situated in the corium or subcutaneous tissue. ey vary in size from a small pea to that of an egg or larger, and are lobulated. They are firm, of about the consistence of adipose tissue, l are movable. The skin over them is normal, although it is stretched l glistening and is usually devoid of hair. The duct of the enlarged nd may remain patulous so that the contents of the tumor can be ressed, but frequently this opening is entirely obliterated and the nor is in a closed capsule. The tumors are usually single, but they y be multiple, and rarely they are numerous. They may occur upon y part of the body, but they are most frequent on the head, espe-lly the scalp, and upon the neck, the back, and the scrotum. They w slowly, and after reaching a certain size may remain stationary years. From their prominence they are likely to be injured, and thus y frequently become inflamed. Occasionally they ulcerate spontane-sly, and in old age they are sometimes the site of development of taneous horns and epitheliomata.

Etiology and Pathology.—Most of them are retention cysts, prob-ly arising from occlusion of the follicular openings. Possibly they are times due to embryonic defects.

Anatomically they consist of a fibrous capsule surrounding a mass of more or less degenerated sebaceous matter, epithelial scales, and *débris*, with perhaps small hairs.

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[1] Crocker, "International Atlas," Plate XXX.—Colcott Fox, *Brit. Jour. Derm.*, 7, vol. ix, p. 21.

Chalazion.—The small, cystic tumors of the Meibomian glands, known as chalazia, are usually classed as retention sebaceous cysts. There seems little doubt that in many instances they are, but according to Virchow, and later Weyman, they are granulomata; Weyman holds that he has demonstrated a pathogenic fungus, the "fungus chalazicus."

Diagnosis.—Sebaceous cysts are liable to be confused with circumscribed lipomata. The chief diagnostic difference is that lipomata are lobulated. Lipomata are also less easily movable, and are oftener multiple and of large size. Upon incision the sebaceous contents of a sebaceous cyst leaves no room for confusion.

Treatment.—They are readily removed by excision. In their removel the excision should be carried down into the cyst and the contents evacuated. Then the capsule is readily dissected out. Unless the capsule is entirely removed the cyst will re-form.

Where the cyst is not large and has a patulous duct opening into it, it is readily treated by pressing out the contents, taking care to see that the cavity is thoroughly emptied, and then swabbing the cavity with tincture of iodine or equal parts of carbolic acid and glycerine. For patulous cysts not larger than a small olive this method is satisfactory. The larger cysts are better excised.

VI.—COMEDO

A comedo is a plug of dried sebaceous matter filling the opening of a sebaceous follicle. The term is applied both to the lesions and to the condition as a whole which they produce.

Comedones are so closely associated with acne that they may be regarded almost as a part of that disease.

Symptomatology.—A comedo is a "black head." It consists of a mass of sebaceous matter and epithelial *débris* filling a more or less dilated sebaceous follicle. Occasionally the sebaceous plug is solid, and can be expressed as an oval, spindle-shaped, hard, semitranslucent mass, but it usually consists chiefly of a mass of semisolid fat which at the mouth of the follicle is capped by a black, hard layer of horn cells and fat. When this mass is pressed out it assumes in passing through the round mouth of the follicle a wormlike shape. Comedones vary greatly in size. In large follicles, as about the nose, they may amount to little more than a normal accumulation of sebaceous matter, or in their extreme size they may be veritable sebaceous cysts. The tips of comedones may either be level with the skin, slightly depressed, or distinctly elevated, giving a nutmeg-grater feel to the skin. They usually are present in numbers, and are symmetrically distributed. They occur especially on the face, the upper part of the back, and to a less degree on the chest. On the face their sites of predilection are the forehead, the nose, and the naso-labial furrows, the angles of the mouth, and the chin, but in extreme cases they may be scattered over the entire face. They are not infrequent upon the vermilion border of the lips. They are chiefly confined to the areas of predilection, but may occur anywhere where there are sebaceous glands. Comedones usually run a more or less chronic course, and in nearly all cases are associated with folliculitis of the sebaceous glands. There may, however, be abundant comedones without any acne, and where acne is present with abundant comedones there are usually numerous comedones showing no inflammation.

Grouped Comedones.[1]—Thin, and subsequently Crocker and Wetherill, have called attention to comedones which occur in groups upon the face, and which are not associated with acne. The affection occurs in densely crowded groups of small comedones, symmetrically distributed upon the face, and usually appearing upon the flush areas. They are apparently a result of reflex flushing of the face, in most cases from dyspepsia.

Large, grouped comedones which show no tendency to produce suppuration, are frequently seen around the outer canthi of old people. The lesions are few and are not to be confused with Thin's affection.

Grouped Comedones in Children.—Crocker has called attention to the occurrence of comedones in children upon parts subject to pressure and warmth. The lesions occur in densely crowded groups, they are round and firm, and occasionally folliculitis occurs around them. They are seen on the cheeks in nursing infants, on the forehead, temples and occiput on older children from friction of the hat band, and occasionally elsewhere. I have seen similar comedones on the forehead from the hat band and around the neck from the friction of the collar band in adults. The condition is rare in the United States, but I have seen a few infants at the breast with the lesions abundantly developed on the cheeks.

Etiology and Pathology.—A comedo consists of a mass of cholesterin and epithelial *débris* surrounded in the upper part by a more or less perfect layer of horny epithelium. The color of the tip is probably due solely to dirt, although Unna attributes it to a pigment, ultramarine, formed from the secretion. Comedones, of course, contain numerous bacteria and very frequently a harmless parasite of the skin, the demodex or acarus folliculorum.

The factors in the production of comedones are multiple. When they are seen in adolescence, the most important factor in all probability is excessive activity of the sebaceous glands doubtless characteristic of that period. In later life the important factors in their production are metabolic and reflex disturbances producing excessive activity of the sebaceous glands. These are for the most part disturbances like those of the gastrointestinal tract producing reflex flushing of the face which are considered under acne rosacea. In addition to the excessive activity of the sebaceous secretion, other factors come into play. Where the general health is lowered and the tissues relaxed, as in anæmia, the glands dilate rather than empty themselves; this is probably due to atony of the arrector pili muscles. Other factors in their production are such as produce mechanical obstruction to the emptying of the follicles, of which hyperkeratosis is the most important. Probably one important factor in many cases where comedones are very abundant is an unusual development of the sebaceous glands. Occasionally individuals are seen whose sebaceous glands are unusually large and in whom, even when comedones are not apparent, long masses of sebaceous matter can readily be pressed out from the larger follicles. At the International Dermatological Congress in London, a case was exhibited with excessive comedones on one side of the back, but the condition was sharply limited by the median line. The condition had persisted for several years, in a young adult, and it was apparently due to excessive congenital development of the glands on the affected side. The theories of the bacterial origin of comedones of Unna, Hodara, and Sabouraud are referred to under Acne.

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[1] Thin, *Lancet*, 1888, vol. ii, p. 712.

The diagnosis, treatment, and further consideration of comedones are taken up with acne.

ACNE

Acne is folliculitis of the sebaceous glands. Its usual form is acne vulgaris, and this is always meant when the term acne is used without qualification. There are, however, other affections in which acne lesions occur, and which are more or less related, clinically at least, to acne. These are:

Acne scrofulosorum,
Acne varioliformis and its congeners.
Acne keratosa,
Acne necrotisans.

VII.—ACNE VULGARIS[1]

Synonyms: *Acne, Acne simplex, Acne disseminata.*

Acne vulgaris, or acne, is characterized by an eruption of inflammatory lesions varying in type from papules to pustules, involving the sebaceous follicles, and in nearly all cases commingled with comedones.

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[1] Touton, *Verhandlungen der Deutschen Dermatologischen Gesellschaft, VI Congress*, 1899, p. 7 (full review and bibliography).—Hodara, *Monats.*, vol. xviii.

The typical picture of acne is the ordinary bad complexion with which everyone is familiar.

Symptomatology.—The picture which acne presents is a varied one, because there may be all variations in abundance of the lesions from a ew discrete inflammatory pustules to an eruption which is so abundant s to disfigure the entire face and upper half of the trunk, and because he lesions vary very widely in the depth of tissue involved. They may be the most superficial pustules simply covered by the horny epidermis, or, on the other hand, they may be small abscesses deep in the orium, which can only be emptied by incision through a thick layer of skin. The characteristic feature of the lesions in all cases is that they nvolve sebaceous or pilo-sebaceous follicles. In most of the lesions a ebaceous plug exists in the center of the lesion; the lesion, indeed, conists of a central comedo surrounded by an inflammatory papule or pusule. In the deeper lesions the comedones may be so disintegrated that upon incision only pus is obtained, but even in these lesions the follicuar opening forms the apex of the lesion, and the commingling of comedones with inflammatory lesions indicates the pathology of the condition.

Clinically, acne may be divided into *acne simplex,* in which the lesions are superficial in the corium, and *deep-seated* or *indurated acne,* in which the lesions occur deep in the corium. Cases occur presenting all grades of the disease between the extreme types.

In the usual case of acne simplex there is an eruption of superficial inflammatory papules and pustules associated with comedones. The pauplar type of lesion may predominate or exist alone (*acne papulosa*), or the pustular type may predominate (*acne pustulosa*); usually lesions of both types are present. The lesions may consist of pustules so superficial that they are easily ruptured in washing with a soft towel. As a rule the lesions are somewhat more deeply seated, and vary in size from a large pinhead to a small pea. Such an acne may consist of only a very few lesions, or the lesions may be so abundant as to pepper the entire surface; and between these extremes there are all degrees of abundance of the eruption. This form of acne is usually associated with more or less oily seborrhœa, further disfiguring the skin by giving it a greasy and dirty look.

In acne indurata, the lesions are very much more deep-seated, and produce greater deformity. In these cases the sebaceous glands form really small sebaceous cysts containing masses of sebum up to the size of a pea or larger, and these glands, becoming infected, form small abscesses

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1894, p. 573.—Sabouraud, *Annales de l'Institut Pasteur*, 1897, p. 134.—Unna, *Histopathology*, 1896, pp. 357–361.—Gilchrist, *Trans. Amer. Derm. Assn.*, 1899, p. 97.—*Jour. Cutan. Dis.*, 1903, p. 107.

in the skin rather than pustules. The lesions occur usually as indolent, but occasionally as acute, small abscesses, which show how the surface as red to purplish tubercles. The pustular contents may show upon the surface as a yellow center of the tubercles, or they may be so deep that this feature is obscured. In this form of acne the lesions are rarely very few. They occur in numbers on the areas of predilection, and in extreme cases they may be so numerous as to literally riddle the skin with small abscesses. In extreme cases of deep-seated, indurated acne, the amount of disfigurement may be very great, and if it is persistent it is a source of great humiliation.

Superficial acne lesions cause no scarring. Unless there is an occasional deep-seated lesion, after the actual disease disappears it leaves no trace. There are rare exceptions to this statement, in which superficial, papular, or pustular acne are followed by minute scars—*acne atrophica*. It is not infrequent, particularly in superficial acnes in nervous girls, to find excoriations over the lesions, as the result of an unrestrained impulse to pick at them or of crude attempts to treat them. In deep-

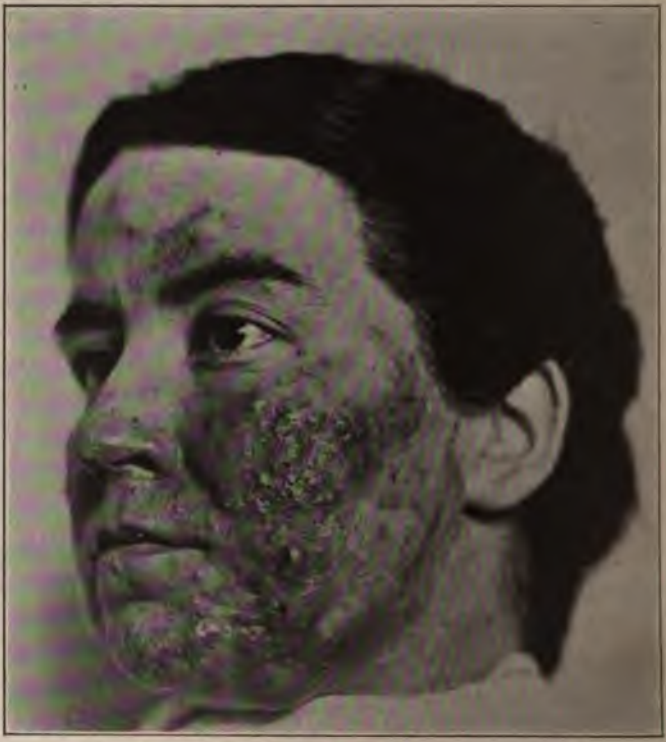

FIG. 330.—ACNE INDURATA. (Hartzell's collection.)

seated acne the follicular abscesses may cause sufficient destruction of tissue to produce distinct atrophic scars. These scars are usually round, obtusely conical depressions, and where the acne has been very severe and long-continued the scarring that results is sufficient to give the skin an irregular pitted surface which is permanent. The scarring from this form of acne, however, consists of smooth, round scars of about normal color, and while the scarring may be as abundant as in severe variola, it is not nearly so disfiguring. In other rare cases of acne, sometimes of the superficial form, but most frequently of the deep type, there are produced scars which are hypertrophic—*acne hypertrophica.* The scars in these cases are rough, irregular, and slightly elevated, and are more noticeable than the usual atrophic scars. This condition is sometimes associated with hypertrophic scarring on the back of the neck, known as dermatitis papillaris capillitii (*q. v.*).

The distribution of acne is characteristic, and corresponds exactly with that of comedones. It is symmetrical, and usually sharply so. The face is the area of predilection, and on the face it is most abundant on the forehead, the nose and the adjacent part of the cheeks, and the chin. Around these areas the eruption scatters in greater or less abundance over the rest of the face and may extend down to some extent upon the neck. Next to the face, the disease most frequently affects the upper half or two thirds of the back, and less frequently the chest. Any one of these areas of predilection may be involved without involving the other two. Frequently one sees a case of severe acne upon the face without any on the back, occasionally the reverse. Superficial acne is likely to be confined to the face with a few lesions upon the back and chest. Indurated acne is more likely to involve the back as abundantly as the face, and acnes confined to the back are usually of severe, indurated type. In addition to the areas of predilection acne may appear to a greater or less extent upon other parts of the body, but rarely involves them to any extreme extent except in artificial acnes.

An individual acne lesion runs a comparatively short course, the superficial lesions disappearing in a few days, the indurated lesions pursuing the course of a small abscess, disappearing quickly after emptying their contents, but lasting from several days up to two or three weeks if indolent and uncared for. The lesions, however, come out without any regularity, and thus tend to continue the disease for months or, in many cases of indurated acne, for years. The moderate case of simple, juvenile acne may appear at puberty or later during adolescence and continue a few weeks to a few months or a year or more, and then disappear of itself. The more severe cases are apt to continue up to nineteen to twenty-one years of age, and may then persist to the age of thirty and beyond. The subjective symptoms of acne are trivial. The superficial lesions are somewhat tender and may itch slightly; deep-seated, follicular abscesses may be quite tender and somewhat painful, but the subjective symptoms are so trivial in comparison with the mental suffering of the patients that they are of no consequence.

Artificial Acne. *Tar and Oil Acnes.*—Occasionally acne is produced by the occlusion of the mouths of the sebaceous follicles by extraneous matter. The commonest illustrations of this are seen in persons whose occupations expose them to contact with tar or fats.

Acne may be produced by the presence of drugs in the circulation, where the lesions are presumably caused by the irritation produced by the drugs in their excretion through the sebaceous follicles. This form of acne is nearly exclusively produced by the bromides and iodides. The lesions are usually large and of indolent character, and develop in the usual areas of acne. These acnes are usually abundant, have rather large lesions, and are peculiar in that they appear upon unusual surfaces.

In tar acne the lesions may involve the usual areas, and in addition occur abundantly upon other parts where clothing impregnated with tar comes in contact with the skin. In machinery oilers and in other individuals whose clothes become impregnated with oil, as in hemp and twine makers, we occasionally see extreme development of acne upon covered parts, like the abdomen and legs, while the face and hands, which are not covered by the greasy clothing, remain entirely free. In acnes of these peculiar distributions an external cause should always be suspected.

Acne cachecticorum.—In rare cases acne, presenting little or no evidence of retention of secretion in the sebaceous glands, develops in cachectic subjects. This form of acne is likely to be seen in cases of extreme anæmia, or in other conditions of malnutrition. The lesions may or may not be indurated, and their peculiar characteristic is the purplish or livid-red color and indolent type. Comedones may be entirely lacking, but in some cases are to be found. The eruption may be confined to the ordinary areas of acne, or may be widely distributed over areas not usually involved, especially the trunk and extremities. The lesions are indolent, pursue a slow course, and are followed by more or less scarring. The condition is closely related if not identical with acne scrofulosorum and folliclis (*q. v.*).

Etiology and Pathology.—Acne is probably the most universal of skin diseases. Including as acne the eruption of the smallest number of acne lesions, there are probably few individuals who during adolescence do not have acne. It is essentially a disease of adolescence and early adult life. It is apt to be worse at puberty and disappear by the age of twenty-one or twenty-two. It rarely occurs before the onset of puberty, but it frequently persists in cases of severe indurated acne to twenty-five years or even beyond. It is no respecter of social classes, and is not evidence of lack of care of the skin. It is not an evidence of irregular sexual functions or habits.

The important factors in the development of acne are conditions which cause the accumulation of sebaceous matter in the follicles. As already seen, the most important cause of this condition is the unusual activity of the pilo-sebaceous glands during adolescence. Other causes are conditions which produce reflex flushing of the face and excessive activity of the sebaceous glands, and conditions of malnutrition which cause atony of the skin. Such conditions may produce acne independent of adolescence, and thus we occasionally see acnes in individuals of twenty-five to thirty-five years or age, or even older. These disturbances of the general health are, however, more frequently accessory factors in exciting acne during adolescence. Among the disturbances of the general health which tend to produce acne are especially digestive disorders and anæmia and chlorosis. The influence of menstrual disorders and uterine disturbances is less frequent, but is occasionally observed. Sexual disorders in males are very rarely a factor.

These factors all act simply to produce comedones; acne then results through the mechanical irritation which they produce combined with infection. According to Unna, Hodara, and Sabouraud, the production of comedones is not dependent upon these physiological disturbances, but upon infection with a micro-bacillus. According to Sabouraud the essential factor in the production of comedones is infection with the specific seborrhœic micro-bacillus. This first, through its entrance into the follicles, produces seborrhœa; accompanying this there is an irritation of the epithelium with the formation in the follicle of a " cocoon " of horny cells which forms a horny plug occluding the follicle, and then to produce the acne secondary infection must occur, according to Sabouraud with the staphylococcus albus butyricus, or with ordinary staphylococci. This theory of the formation of comedones has not obtained general acceptance, and does not square well with the general characteristics of the affection. The production of artificial acne lesions occurs either by irritation of the follicles or by mechanical plugging. In acne due to the ingestion of bromides or iodides this irritation is produced from within. In tar acne it is probable that the effect is not so much from the mechanical plugging of the follicles by tar as from the irritation caused by tar. This is more plausible from the fact that other local irritants, like chlorine and chrysarobin, may at times produce acne. In acnes produced by long contact of greasy materials, like oils, fats, and paraffin, mechanical plugging of the follicles is the cause.

The pathology of acne includes the consideration of the formation of comedones, and the inflammatory changes which follow. The formation of comedones has already been considered. After the development of comedones, there occur inflammatory processes from infection around the follicles resulting in an infiltration with leucocytes, combined with plasma, lymphoid, new connective tissue and giant cells. The neighboring blood vessels are dilated and there is a perivascular leucocytic infiltration. The process may stop at this point, resulting in the formation of inflammatory papules; or the leucocytic infiltration may increase and undergo neucrosis with the formation of pustules or abscesses. According to the depth at which the inflammation occurs or to which it extends rests the distinction between superficial acne, or acne simplex, and deep acne, or acne indurata.

The bacteriology of acne is unsettled. Sabouraud's theory of the rôle of his micro-bacillus in producing comedones has already been referred to. He holds that the subsequent inflammatory stage is due to secondary infection with the staphylococcus albus butyricus. Gilchrist, who has studied this question extensively, has described a bacillus which he calls the bacillus acnes, and which he thinks is a specific cause of acne. He has obtained the organism in pure cultures from 62 lesions, and in smears from 240 lesions; in other cases the organisms occurred with the staphylococcus albus. The bacillus acnes is a short, thick, slightly curved bacillus which often becomes longer and thicker in cultures, and in old cultures develops branching forms. The organism is slightly motile, noncapsulated and stains by Gram's method. It grows on practically all media; on agar as a pure white pultaceous mass, elevated, moist, smooth, and glossy. It is pathogenic for mice and guinea pigs, and is agglutinated by the patient's serum in dilutions up to 1:100.

Gilchrist reverses the usual order of things, and suggests the highly novel idea that the constitutional symptoms so often found in acne, like anæmia, headache, and constipation, are the results of intoxication by poisons produced by the specific bacilli, rather than predisposing causes of the disease. The fact that the bacilli are agglutinated by the patient's blood lends great weight to his position.

The generally accepted view is that the inflammation in acne is produced by infection with the common pyogenic organisms, usually the staphylococcus albus. Certainly the infection of comedones with the staphylococcus albus can produce acne lesions, and it is quite possible that we have acne of two forms, one due to staphylococcus infection, the other due to infection with the bacillus acnes, both resulting in the same clinical pictures.

Diagnosis.—The characteristic features of acne are first the manifest follicular character of the lesions, and the association with comedones. Other features are its characteristic disturbances, the age of the patient, and its slow course. The disease can rarely be mistaken for anything else. The two affections with which at times it may be confused are a papulo-pustular syphilide and variola. The clinical features of the papulo-pustular syphilide are sufficiently characteristic to make a positive diagnosis. The papulo-pustular syphilides are not so manifestly associated with the follicles of the skin and comedones. They are especially abundant about the nose and mouth and forehead. They tend to circinate grouping. They undergo central necrosis, with a wider area of ulceration and without the central conical abscess of the acne pustule, and they produce larger variolalike scars. Most important of all in the diagnosis for the inexpert is the fact that the pustular syphiloderm is never confined to the acne areas, but has a wider distribution, and is associated with other lesions of syphilis, especially mucous patches and general adenopathy, and often condylomata. Of course the constitutional symptoms and the history are important evidences, but these may be lacking.

A discrete, mild variola may be at times difficult to distinguish from acne by the appearance of the lesions alone. The lesions of variola, however, are larger, and have an aggressive, prominent character which is peculiar. They are apt to be present on the hands, and of course they are of acute development. The history of the prodromal symptoms of smallpox, particularly in the absence of a good vaccination mark, is also of great importance in making the diagnosis.

Prognosis and Treatment.—The constitutional treatment of acne is confined to meeting rational indications. The most important points requiring attention usually are digestive disturbances and the diet. Constipation in girls is especially a common occurrence in the disease, and is treated by the usual methods. One prescription which is deserving of special mention is the much used "mistura ferri acida":

℞ Iron sulphate	gr. i–ij;
Magnesium sulphate	ʒij;
Dilute sulphuric acid	♏xv–xxx;
Peppermint water	℥i.

Dose, a tablespoonful in a glass of water one half hour before breakfast.

Tonics of iron, strychnine, and arsenic are often indicated in anæmic and debilitated subjects, and arsenic, I believe, is often beneficial for its specific effect upon the skin. The diet should be looked to carefully, and the patients placed upon a simple, nutritious regimen. Aggravations of acne can at times be definitely traced to indulgence in sweets and pastries and highly seasoned made dishes, and these are to be avoided. Avoidance of butter and fats, so often arbitrarily ordered, is not necessary. Of course hygienic living, plenty of fresh air and sunshine and exercise are desirable, particularly in depleted patients.

Aside from the doubtful benefit derived from arsenic, the specific remedies suggested in acne, like calcium sulphide, ichthyol, and sulphur are in my opinion useless, although sulphur in 5-grain doses three times a day may be a useful laxative. Where there are definite, rational indications to be met in the physical conditions of acne patients, constitutional treatment is of benefit; it is particularly necessary to get rid of constipation and other digestive disturbances. But on the whole the relief of acne depends upon local treatment.

The indications to be met in the local treatment of acne are: to cause exfoliation of the thickened horny epidermis, to diminish the excessive fat secretion and overcome the formation of comedones, to sterilize as far as possible the skin, and to get rid of the products of suppuration. All of these indications are met to a greater or less extent by the use of certain keratolytic and astringent antiseptics, like sulphur and some of its salts, corrosive sublimate, and resorcin. Of these the sulphur preparations are the most valuable.

In the first place the patient should be instructed to keep the skin clean by the use of soap containing some free alkali, which will get rid of the grease and by its keratolytic action cause exfoliation of the thickened horny epidermis. Tincture of green soap is best for this purpose. In thick greasy skins an ordinary laundry soap may be used. The parts should be thoroughly washed with soap and warm water for several minutes at night, and this should be followed by rinsing in cold water. Comedones should be removed and pustules opened at intervals of a few days by the physician. The comedones can be removed by the pressure of clean thumb nails or by the use of a comedo extractor, or, better still, by pressure with the back of a broad dermal curette. The pustules are most conveniently opened with a small iris knife. After they are opened the contents should be evacuated with as little pressure as possible, and each lesion touched with carbolic acid applied on a fine probe. Where the lesions are superficial, especially if the comedones project above the surface, the same results can be obtained by curetting the tips of the lesions, after which their contents are pressed out and the surface washed in warm water, or a mild antiseptic solution. The end attained by such local treatment is not only the cleaning of the skin, but its stimulation, and occasionally further stimulating measures may be useful, such as massage, cupping, the application of a faradic or galvanic or high-frequency current. These measures are, however, in my experience of little use.

At the same time astringent keratolytic antiseptic applications are used. The best of these are lotio alba and Vleminckx's solution. At the beginning the patient should use Vleminckx's solution in about 1 to 10 parts of water. After washing the face at night he should athe it with this for five to ten minutes. The bathing is done with more r less vigor until slight irritation is produced. The solution should e allowed to dry upon the face, and in the morning the face should be insed in soap and water. The application should be continued nightly ith sufficient vigor to keep just short of chapping the skin. After four r five days the strength of the wash should be increased to 1 to 9, and o on as long as tolerance lasts. As a rule the strength can be increased radually up to 1 to 5 or 1 to 4. Occasionally it is necessary to produce oderate dermatitis in order to get rid of the acne. When dermatitis ccurs the application should be stopped and bland ointments used ntil the reaction subsides. The vigor of the treatment should be egulated according to the variety of acne and the tolerance of the dividual skin.

Essentially the same plan of treatment can be carried out by the se of lotio alba. The patient should start with a lotion containing rom 3 to 5 grains of potassium sulphide and zinc sulphate to 1 ounce f water, and gradually increase to 15 grains. Lotio alba may be sed frequently—two or three times daily—but the treatment is carried ut essentially in the same manner as with Vleminckx's solution. Either f these methods of treatment is usually effective in rapidly cleaning up n acne. They, however, must be persistently carried out and repeated rom time to time as recurrences develop.

Among other agents which may be used in place of sulphur, corrosive ublimate and resorcin are especially to be mentioned. Resorcin may be sed in solutions of from one- to ten-per-cent strengths. With the sulhur and resorcin washes, soap containing sulphur and resorcin, such s are commercially obtainable, may be used, but they offer little or no dvantage over unmedicated soaps.

Bichloride solution in strengths of 1:2,000 to 1:500 is a useful pplication, especially in milder acnes. It may be combined with a ichloride soap, and the two are used just short of producing dermatitis, s in the methods already outlined. In shifting from sulphur applicaions to mercurials, care should be used to get all of the sulphur off the kin before the mercurial application is used; otherwise numerous comeones are produced by the formation of mercuric sulphide at the mouths f the follicles.

In very severe acnes occasionally great benefit is produced by the use f strong paste which produces acute dermatitis and abundant scaling of he skin. A paste of this sort is fifteen- to twenty-per-cent resorcin in inc ointment. This is applied at night until burning begins—for twenty o thirty minutes—and is then removed by wiping off with vaseline or live oil. This is continued nightly, slightly increasing the length of the pplication each time until dermatitis is excited, which is treated by the application of cold cream or other bland ointments. A still more vigorous scaling application is a paste suggested by Lassar:

℞ Beta naphthol	gr. l;
Precipitated sulphur	ʒiv;
Rose ointment, green soap, each	ʒij.

This is applied at night for from fifteen to thirty minutes, then wiped off with olive oil, and the surface washed with water and powdered with a bland powder. After a few applications it produces an acute dermatitis with free desquamation, which, however, subsides in the course of a few days under bland applications like rose ointment. Either of these methods produces rapid improvement, but they have the disadvantage of producing acute disturbance, and they also require occasional repetition.

With the various methods of local treatment given above great temporary improvement can be produced in acne, and in most cases satisfactory results can be obtained. A permanent result is much more likely to occur if the condition is largely dependent upon disturbances of the general health which can be corrected. It is a fact, however, that in many cases these methods fall far short of radical relief.

In the last few years a great deal has been done in the treatment of acne by the use of x-rays, and the method is used largely by dermatologists at the present time.[1] The rationale of its use depends upon the fact that by x-ray exposures the functional activity of the sebaceous glands can be diminished. In carrying out the treatment, short mild exposures are given at intervals of several days, care being taken to avoid completely any evident reaction in the skin. My method of treatment is to give exposures of five minutes' duration with the wall of the tube at a distance of six inches from the surface and with only enough x-rays to produce a faint green glow in the tube. These exposures are given from one to three times per week, and they are continued until the desired improvement is obtained. It is not necessary to produce any apparent reaction, and the exposures should not be given with sufficient vigor to render a dermatitis possible. Under the treatment seborrhœa disappears first; the formation of comedones and acne lesions gradually ceases; and on account of the diminution in size of the sebaceous follicles, the pores becomes small, and the texture of the skin much improved. In many cases the result is practically permanent, the patient afterwards never having more than an occasional isolated lesion. In other cases the treatment has to be repeated for a short time at intervals of several months. The treatment is, in my opinion, the method of preference in the treatment of acne, but it should not be undertaken except under conditions of safety. If too great x-ray effect is produced there occurs more or less atrophy of the skin shown by wrinkling, and if moderate burns are produced they may be followed by atrophy and the formation of telangiectases.

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[1] Pusey, *Jour. Cutan. Dis.*, May, 1902.

While giving x-ray exposures in acne, it is necessary to avoid the use of irritating applications, because the dermatitis which they may produce may give rise to confusion with x-ray dermatitis, but the washing of the face and the opening of pustules and removal of comedones can be carried out as usual. These accessory measures, however, are not necessary to successful results from the use of x-rays.

High-frequency currents, faradism, and galvanism are all recommended in the treatment of acne. Aside from the effect resulting from the mild stimulation of the skin which they produce, which can be more readily obtained from the usual irritating applications, these agents are in my experience of no value.

Treatment by injections of sterile dead cultures of staphylococcus albus or of sterile dead cultures of the organism grown from the acne lesions, after Wright's method, is often effective, and should be tried in intractable severe cases. It may give brilliant results; at times also it fails.

VIII.—ACNE ROSACEA[1]

SYNONYMS: *Rosacea, Acne erythematosa.*

Acne rosacea is a disease of the flush area of the face, characterized by permanent congestion, and the formation of telangiectases and acne lesions.

Rosacea is the familiar red nose of dyspeptics and alcoholics.

Symptomatology.—The affection is an angioneurotic disturbance, and is essentially a chronic, reflex flushing of the face. In its first and slightest manifestation there is a slight, diffuse redness of the nose, particularly of the lower half. This varies in intensity, flushing up with attacks of indigestion, with indiscretions in diet, or the use of alcoholics, or from exposure to cold or heat; but with variations like this the hyperæmia is persistent. The nose is pink to dull red, and is cool to the touch. Sooner or later telangiectases develop. These usually first appear crossing the naso-labial folds, but they also develop over the nose and to a greater or less extent on the adjacent parts of the cheeks. At the same time there is some dilatation of the sebaceous follicles. The pores become patulous, and the nose is greasy and shiny. These symptoms may be confined to the nose, and the affection may not pass beyond this stage. In the severer cases, however, the condition becomes intensified and spreads to other parts of the flush area of the face—the cheeks, the center of the forehead, and the chin. As the affection progresses, the redness becomes more marked, perhaps with a purplish tinge. The dilated capillaries become more abundant, the sebaceous glands increase in size, and discrete acne lesions develop in the involved areas. The picture is then one of a greasy, red, congested dermatitis, with indolent follicular abscesses dotting the surface and telangiectases crossing it to a greater or less extent.

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[1] Unna, *Trans. 6th Inter. Derm. Cong.*, p. 78.

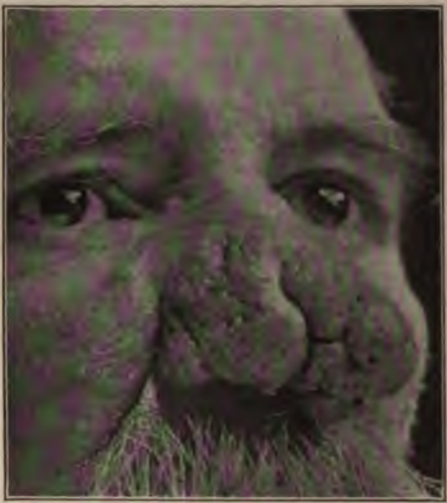
FIG. 331.—RHINOPHYMA. (Grover W. Wende's collection.)

As a rule, the disease does not pass this stage, but in some cases connective-tissue hypertrophy takes place, accompanied by great dilatation of the sebaceous follicles. This is usually limited to the lower half of the nose. In most cases this does not pass beyond the condition of an irregular thickening of the skin with the formation of very large sebaceous follicles, but in occasional cases it goes on until the tip of the nose is converted into a lobulated, tomatolike mass which is pitted all over by the conical openings of enormously dilated sebaceous follicles. Usually these hypertrophic masses are red to purplish in color, but the color may remain a faint pinkish white. This condition, known as *hypertrophic rosacea* or *rhinophyma*, may or may not be associated with rosacea of the rest of the face. Rosacea is persistent, with little tendency to disappear unless the underlying causes are removed. Rhinophyma is a permanent hypertrophy. Neither rosacea nor rhinophyma has any subjective symptoms.

Etiology and Pathology.—Rosacea in its milder manifestations is not infrequent. It is commoner in women than in men, and usually develops after the age of thirty, but in very rare instances it occurs much earlier. The severer forms are more frequent in men and in those whose occupations require exposure to the weather and who are of irregular habits. The exciting factors of rosacea are those which tend to produce reflex flushing of the face. Of these the most important are digestive disturbances. So true is this that in practically every case one can predicate that there is a disturbance of digestion, usually accompanied by constipation. Another factor in women, but in my opinion a relatively unimportant one, is disturbance of the genital tract. In many cases,

ırly of the severer type, the habitual use of alcoholics is the essen-
se of the disease. Alcohol acts in a double way in producing
It produces chronic gastritis and other digestive disturbances,
ılso directly causes flushing of the face. In those who already
ıacea, the use of very hot foods and drinks also acts directly by
ıg flushing of the face. In justice to the unhappy victims of
it should be emphasized that intemperance in drinking or in
; by no means necessary to the production of the disease.

ıophyma usually occurs in alcoholics. I have, however, had an
case of rhinophyma in an old man who had always been a total
r. It may in some cases be due to local conditions which produce
ıia. According to Seiler and others it may result from intra-
sions which produce vascular obstruction. According to Elliot
'isch it may result from the constant hyperæmia produced by
tis of the hair follicles in the nostrils. When the constitutional
ns exist which produce ro-
xternal causes may be im-
exciting factors. This is
ed by the frequency of the
n in cabmen, who combine
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also that lack of cleanli-
l the use of cosmetics and
other sources of local irri-
ıay be contributory factors
ing the disease, but of this
n doubt. Unna and his
ıake the position that the
n is essentially a local one,
manifestation of an infec-
h the ubiquitous seborrhœic
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es, hypertrophy of the sebaceous glands, and the formation of
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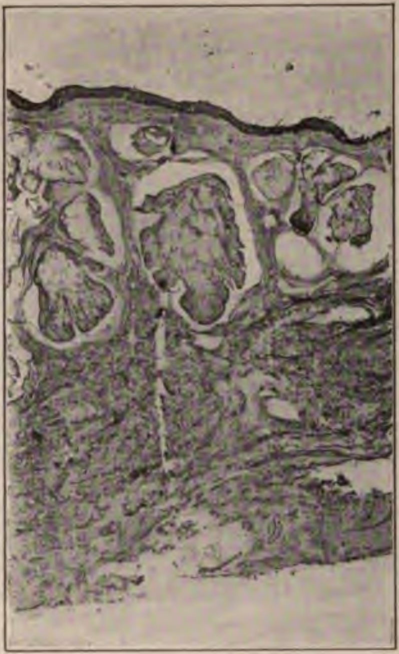

Fig. 332.—Microphotograph, Acne Rosacea. (C. J. White's collection.)

gnosis.—Acne rosacea is sufficiently characteristic to prevent
n in diagnosis; indeed, the patients make the diagnosis them-

selves. From simple acne it is diagnosticated by the diffuse redness which accompanies it, its limitation to the flush area of the face, the presence of dilated capillaries, and the age of the patient. From ulcerative tubercular syphilides of the nose it is differentiated by its symmetry, the absence of destructive ulceration and of characteristic scarring and of the several other characteristic features of tubercular syphilides. Seborrhœic eczema in the naso-labial furrows, accompanied by oily seborrhœa of the nose, which has been referred to in the consideration of seborrhœa, produces a seborrhœic rosacea which must be distinguished from true rosacea. In this, the redness is a faint pink, the enlargement of the follicles is less, there is no dilatation of the capillaries unless true rosacea also exists, and there may be present areas of superficial dermatitis covered by greasy scales.

Prognosis and Treatment.—The radical cure of rosacea is largely dependent upon removal of the underlying causes, and is often impossible. Much, however, can be done to improve the condition. In the first place, attention should be paid to the general health of the patients. Digestive disorders should be properly treated, and special attention given to the regulation of the bowels, and to the avoidance of unsuitable foods. Highly seasoned foods and very hot foods and drinks should be especially avoided on account of their direct effect in causing flushing of the face, and the use of alcohol should be entirely stopped.

The local treatment is very like that of acne. The face should be kept clean by the use of soap and water, but hot or cold water should be avoided and the bathing done with tepid water. Pustules should be opened and treated in the same way as in acne. Much can be done in preventing the formation of pustules and in reducing the redness by the use of strong astringent applications similar to those used in acne. The best of these is lotio alba, which should be used in sufficient strength to cause some irritation and some desquamation.[1] It should be used in the strength of 5 to 15 or 20 grains of potassium sulphide and of zinc sulphate to 1 ounce of water, and should be employed frequently enough to keep the surface dry and slightly desquamating. Vleminckx's solution may be used in the same way as in acne. Strong lotions of resorcin also may be used, but in my experience they are not nearly so satisfactory as the sulphur applications. By the intelligent use of lotio alba, indeed, all that is possible from local medical applications can be done. Most or all of the redness and folliculitis can be gotten rid of, but recurrences are likely to occur and treatment must be repeated.

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[1] Bruck (*Monatshefte* xxxv, p. 275), recommends, for the removal of the shine and redness in rosacea, the application of benzine. It is a solvent of the grease and its volatility causes blanching of the skin. Deodorized benzine is the best preparation. It is used by applying on compresses for a few seconds.

The dilated blood vessels are permanent unless they are destroyed by local means. This is best done by electrolysis, carried out in the same way as in the treatment of other telangiectases.

The connective-tissue hyperplasia can only be gotten rid of by mechanical means. When it is slight, great improvement can be produced by multiple scarification or by multiple puncture, frequently repeated. In rhinophyma the masses can be successfully treated by paring them off. Healing is prompt with little scaring, and the results are satisfactory.

X-rays in the severer cases of acne rosacea offer the best means of permanent improvement. The acne and the seborrhœa can be gotten rid of and more or less atrophy of the sebaceous glands produced. The results are persistent, and the improvement from the elimination of these features of the affection is very pronounced. There may also be obtained considerable improvement in the diffuse redness, which can be carried still further by the use of the usual astringent applications. X-rays produce no change in the telangiectases, but there may be an appreciable diminution in the connective-tissue hypertrophy.

ACNE VARIOLIFORMIS AND ALLIED AFFECTIONS

Synonyms: *Tuberculides, Paratuberculosis.*

There remains a group of rather rare dermatoses, characterized by acnelike lesions, and most of them designated by titles including the name acne, whose nosological position is uncertain. They overlap each other in their clinical manifestations so that sharp differentiations between them cannot always be made. Indeed, in the present state of our knowledge we can only consider the clinical types, and must wait for fuller observations before they are definitely grouped. The individual lesions present similar characteristics. They are for the most part indolent inflammatory papules, sometimes in the pilo-sebaceous follicles, sometimes independent of the follicles. They become pustular or rarely vesicular with necrosis of the apex, and are followed by slight scarring. All of the affections pursue a chronic, indolent course, and the methods for treatment of them are the same. They differ chiefly in the distribution of the lesions and in differences in etiology. As a rule they are only troublesome because of the scarring which follows, and are practically without subjective symptoms. Among these affections are:

Acne varioliformis
Acne keratosa
Acne scrofulosorum
Acnitis
Folliclis
Acne urticata
Acne exulcerans serpiginosa nasi of Kaposi

Each of these has various synonyms, as different authors have independently described cases and given them individual names according to their clinical appearances or the author's views of their pathology. Acne varioliformis may be taken as the most sharply defined affection in the group, and the one under which probably most of them might be grouped.

Acne scrofulosorum, folliclis, and acnitis are for the most part paratubercular affections. The others cannot be grouped according to any definite common etiology, although they usually occur in individuals not in vigorous health, frequently in the tuberculous and in those with feeble peripheral circulation.

IX.—ACNE VARIOLIFORMIS[1]

Synonyms: *Acne rodens* (Vidal and Leloir), *Acne necrotica* (Boeck), *Lupoid acne*, *Acné à cicatrices deprimée* (Besnier and Doyon), *Acne frontalis*, *Acne atrophica*.

Symptomatology.—The lesion of acne varioliformis begins as a small, rounded, superficial, inflammatory papule with a central cap of horny epithelium. As this enlarges there develops upon the tip a vesicle with sero-purulent contents or a thin pustule. This dries into a crust, and the lesion undergoes slow involution with the formation of a superficial, slightly atrophic, pitted scar like that after a vaccination pustule. The peculiar characteristic of acne varioliformis, and of the allied affections, is this atrophic, ultimately white scarring. The lesions on their full development are indolent, flat papules from the size of a pin's head to a pea, and the scars which they leave show similar variations in size. The lesions occur around the sebaceous follicles, and are often pierced by hairs. They may be irregularly scattered or grouped. When they occur in groups, confluent scars may follow which are indistinguishable from good vaccination scars. The eruption occurs characteristically on the forehead and temples, particularly at the hairy border. They also occur on the scalp, and less frequently they appear upon other parts of the face and on the neck. They may develop on the chest and back. In a case which has long been under my observation, the lesions were limited to the shoulders and the upper third of the back. The number of active lesions which are present at one time is usually few (from a half dozen to a score or more), but from the scarring which results the disease may in time produce very marked changes in the appearance in the invaded areas. The individual lesions are indolent, lasting from one to three or four weeks, and the disease as a whole is chronic, continuing for months or years with outcropping of a few lesions at a time. The eruption is usually accompanied by oily seborrhœa.

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[1] Bronson, *Jour. Cutan. Dis.*, 1891, p. 122.—Fordyce, *Jour. Cutan. Dis.*, 1891, p. 128.—*Jour. Cutan. Dis.*, 1894, p. 152.—Johnston, *Phila. Med. Jour.*, February, 1899.—Bronson, *Jour. Cutan. Dis.*, 1905, p. 121.

Etiology and Pathology.—The disease occurs in men and women, and usually in those over thirty. Fordyce suggests that lack of cleanliness of the skin, with resulting infection with staphylococci, may be the etiology of the disease, but the disease is not confined to the uncleanly. Johnston regards it, upon apparently good grounds, as a toxic disturbance and one of the paratuberculoses, along with folliclis, acnitis, and acne scrofulosorum. Anatomically the lesions begin as a horny plug in the hair follicles, followed by inflammation and necrosis. The sebaceous glands may or may not be involved. Pyogenic staphylococci are found in

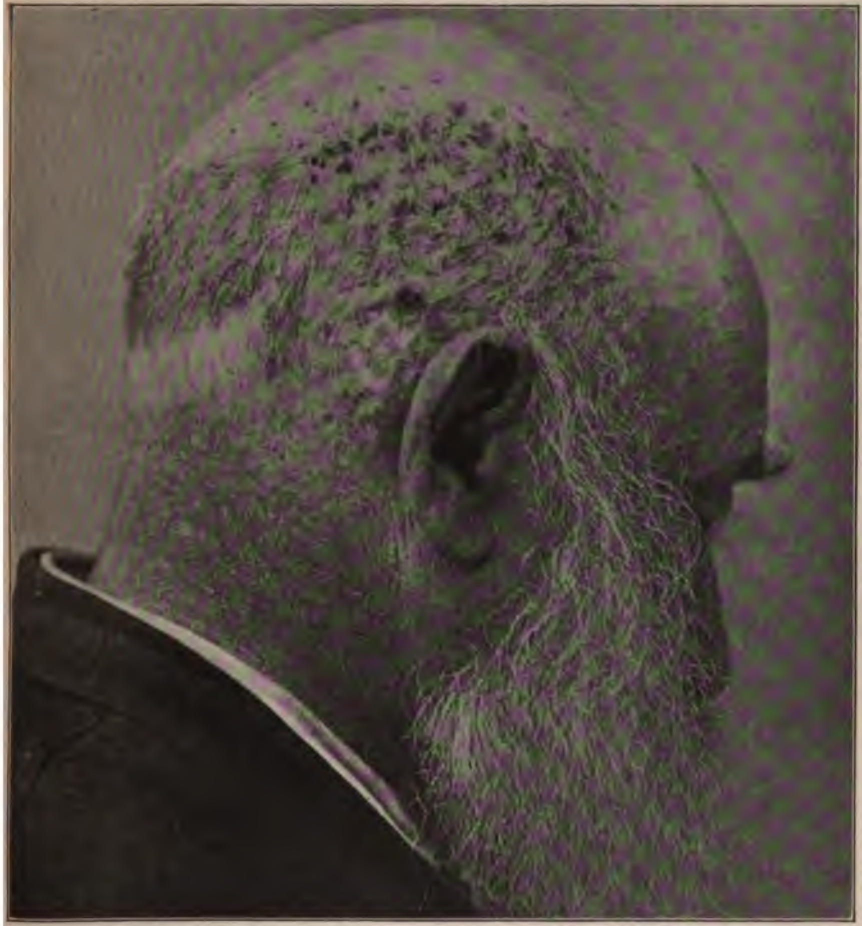

FIG. 333.—ACNE VARIOLIFORMIS. (Fordyce's collection.)

the lesions, but their presence is probably secondary to the irritation of horny plugs which are the essential anatomical feature of the disease. Sabouraud regards the horny plugs as due to his seborrhœic micro-bacillus, just as comedones are, and attributes the affection to the specific action of that organism.

Treatment.—The local treatment of acne varioliformis consists in the use of antiseptics, of which the mercurial applications are the best. The parts are to be kept sterilized by bathing with bichloride solution or some other antiseptic solution, after which the lesions may be touched with carbolic acid. Where intestinal autointoxication is suspected, salol is recommended, and cod-liver oil is of benefit in the strumous cases. Iron is highly recommended as a tonic in the cases. Arsenic may directly cause improvement in the skin. Crocker believes that the cases are improved by potassium iodide internally. Relief, however, has to be obtained from local treatment, and by it temporary disappearance of the disease can be brought about, but the eruption is likely to recur.

X.—ACNE KERATOSA[1]

Crocker has described five cases of a peculiar folliculitis occurring in the mouths of the hair follicles, in which the peculiar characteristic is the existence in the lesions of soft or horny, conical plugs about one twelfth of an inch long, embedded in the skin, and causing the development of inflammatory lesions which the patient excoriates. The lesions are superficial inflammatory papules and undergo involution with scarring. They appear singly or in small numbers, and on account of the excoriations usually are covered with blood-stained crusts. They are symmetrically distributed, with the cheeks and chin as the areas of predilection.

Four of the cases were women, the fifth a man. Jamieson regarded the plugs as comedones, but in Crocker's opinion they are made up of horn cells derived from the hair follicles. These horny pegs, like comedones, produce the mechanical irritation which is the primary cause of folliculitis.

XI.—ACNE SCROFULOSORUM

This is an affection of tuberculous children, consisting of minute papulo-pustules which occur in groups on the extremities and the lower part of the trunk. The lesions are of the size of a pinhead or larger, very rarely reaching the size of a small pea, are situated around the hair es, and are usually small pustules on a red or purplish-red base. show marked tendency to appear in small clusters, and the eruption ally abundant. The lesions come out in groups, and on disappearing purplish stains and very rarely superficial scars. The disease as a is chronic, tending to improve or disappear with improvement in ıtient's health, and to recur with exacerbations of tuberculosis.

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[1] Crocker, "Acne Keratosa," *Brit. Jour. Derm.*, 1899, vol. xi, p. 1.—*Monats.*, vol. xxxiii, p. 405.—Gottheil, *Amer. Med. Assn. Jour.*, July, 1904.—Finger, *Wien. klin.-therap. Wochenschr.*, 1904, No. 41.

occurs in young children, occasionally in adolescents, and usually hectic patients with foci of tuberculosis. Histologically the lesions ıdolent inflammatory infiltrations around the hair follicles, not ng the structure of lesions of tuberculosis. They do not contain , and inoculation experiments have been negative. The clinical cteristics of the condition are sufficiently clear, in association with ulosis or a tuberculous cachexia, to render the diagnosis easy. pare acne cachecticorum.)

ıe treatment consists in the internal use of cod-liver oil and in the al measures useful in tuberculosis, and of local antiseptic applica- An ointment of sulphur or resorcin, two or three per cent, applied daily usually removes the lesions, but they recur unless the toxæmia gotten rid of.

XII.—ACNITIS[1]

(Barthélemy)

NONYMS: *Acne agminata, Disseminated Follicular Lupus, Small Pustular Scrofuloderm* (Duhring).

acnitis the lesions are ed, indolent, inflamma- papules which may or ıot develop around come- They become purulent, e an indolent course, and followed by superficial ng. The characteristic e is the grouping of the s on the face—on the the central areas of the , in the eyebrows, and glabella, and over the es. In some cases a esions are found on the

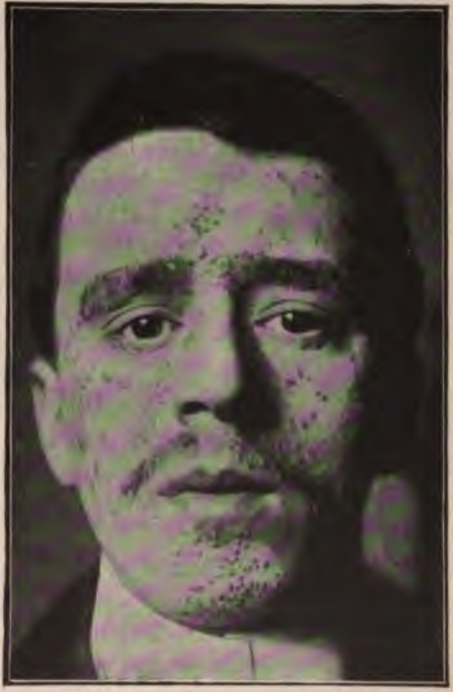

FIG. 334.—ACNITIS. (Crocker.)

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[1] Barthélemy, *Annales de Derm.*, vol. ii, 1891, p. 1.

forearms, and lesions may occur on the mucous membranes of the mouth.

The association of the disease with a tuberculous cachexia or a tuberculous family history is frequent, and has suggested that the condition is a paratuberculosis resulting from tuberculous toxins. Barthélemy attributes it to autointoxication from the gastrointestinal tract.

Anatomically the lesions consist of dense infiltrations around the pilosebaceous follicles, the structure in general suggesting that of tuberculosis, but tubercle bacilli do not occur in the lesions.

XIII.—FOLLICLIS[1]

Synonyms: *Acrodermatitis pustulosa hiemalis* (Crocker), *Lupus erythematosus disséminé*, *Folliculites disséminées symétriques des parties glabres à tendance cicatricielle*, *Acne varioliformis of the extremities* (Bronson), *Folliculitis exulcerans* (Lukasiewicz), *Hidradenitis destruens suppurativa* (Pollitzer), *Hydrosadénite suppurative disséminée* (Dubreuilh), *Scrofulides nodulaires disséminées* (Dubreuilh), *Spiradenitis suppurativa disseminata* (Unna), *Tuberculides* (Darier), *Granulome innominé* (Tenneson), *Folliculites tuberculeuses*, *Tuberculides acneiformes et nécrotiques* (Hallopeau, Balzer, and Leroy), *Toxituberculides papulo-nécrotiques* (Hallopeau), *Acne cachecticorum* (?).

Folliclis occurs as indolent papulo-necrotic lesions, occurring especially on the extremities—the hands, feet, forearms, and legs—but the face may also be attacked. For the most part, the lesions pursue a more acute course than in acnitis, but they are indolent, inflammatory papules, and the disease as a whole lasts for years.

Symptomatology.—As indicated by the numerous synonyms given above, the characteristic feature of the disease is the tendency to superficial scarring, and the fact that such observers as Bronson and Isaac have reported cases as acne varioliformis of the extremities, is highly significant of the similarity of the disease to acne varioliformis except in distribution. Minor differences in the lesions of acnitis and folliclis are described by Barthélemy and others who would separate the diseases, but unless the affections can be shown to have more essential differences than are now known, they may be regarded as differing only in distribution: this is the position held by most of the French dermatologists, to whom we are particularly indebted for the study of these affections.

Etiology and Pathology.—Most of the cases occur in individuals with a personal or family tuberculous history, or in those with weak peripheral circulation. The histology is in dispute. Pollitzer, Unna, Fordyce, and others regard the disease as a sweat-gland folliculitis

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[1] Johnston, *Phila. Med. Jour.*, February 1, 1899.—Barthélemy, *Annales de Derm.*, vol. ii, 1891, p. 1.—Bronson, *Jour. Cutan. Dis.*, 1891, p. 121; Colcott Fox, *Brit. Jour. Derm.*, 1900, p. 383.—Hartzell, *Medical Record*, June 23, 1906.

(hydradenitis), while others regard it as a granuloma independent of the follicles.

Small Pustular Scrofuloderm of Duhring.—Duhring[1] has described a small, pustular scrofuloderm, occurring in scrofulous subjects, with lesions on the face and upper extremities like those of acne varioliformis. In its distribution it forms a connecting link between folliclis and acnitis.

Diagnosis of Acne Varioliformis, Acnitis, and Folliclis.—Acne varioliformis, acnitis, and folliclis may possibly be confused with papulo-pustular syphilides. Aside from the characteristic clinical features of the papulo-pustular syphilide and the abundance of the lesions, the absence of other features of active syphilis will make the diagnosis easy.

From acne these affections are distinguished by the absence of manifest association with the comedones, by their indolent course, and especially by their characteristic, atrophic scarring. From each other they are distinguished chiefly in distribution.

Acnitis and folliclis are treated in the same way as acne varioliformis.

XIV.—LICHEN NITIDUS[2]

In the Neisser Festschrift, Pinkus has described under the name lichen nitidus a peculiar eruption occurring about the genitals. The eruption consisted of numerous, discrete, pin-head size papules of the color of the skin. The lesions were devoid of evidence of inflammation and resembled somewhat small smooth glistening warts and were without subjective symptoms. Histologically the lesions were sharply defined granulomata made up of epithelioid and giant cells. Pinkus described nine cases, all of them confined to the genitals. Kyrle and McDonagh have reported a case in a girl, showing the same lesions, but occurring as a generalized eruption. The disease occurred as a papular eruption without inflammatory symptoms, involving the flexures, especially the wrists and elbows, the front and sides of the thorax, the abdomen and the genitals. As in Pinkus's cases the papules were flat or conical and had a waxy, glazed appearance. Histologically this case was identical with Pinkus's cases. Kyrle and McDonagh's case showed a marked tuberculin reaction. Kyrle and McDonagh, from the histological appearance, believe that the condition must relate to the tuberculous process, and the condition as a whole suggests that the disease falls within the group of the tuberculides.

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[1] Duhring, *Amer. Jour. Med. Sci.*, 1882, vol. lxxxiii, p. 70.

[2] Pinkus, *Archiv*, 1907, B. 95, p. 11.—Kyrle and McDonagh, *Brit. Jour. Derm.*, 1909, p. 339.

The disease is distinguished from lichen planus by the transparent appearance of the lesions, typical of granulomata, the absence of redness, and the fact that the papules have no tendency to increase in size and undergo no change in color. The histological pictures are, of course, entirely different. From lichen scrofulosorum it is distinguished by the non-follicular character of the lesions and the entire absence of scaling.

C. DISEASES OF THE HAIR[1]

I.—LEPOTHRIX

SYNONYMS: *Trichomycosis nodosa, Trichomycosis palmellina.* ETYMOLOGY: λεπίς, scale; θρίξ, hair.

Lepothrix is the term applied to concretions upon hairs produced by the growth of microörganisms. They may occur as discrete masses at the sides of or surrounding hair shafts, or as continuous masses enveloping them completely. They are usually reddish, and are often associated with red sweat, but they may be of darker to blackish color. The organisms producing them may or may not invade the hair shaft, but where the hair shaft is invaded the cortex is broken up. The hairs are made brittle by the concretions, and may break either with a clean fracture or by brushlike splitting of the shaft.

The concretions consist of microörganisms, and a hard, homogeneous cement substance which they elaborate. The condition is probably produced by several organisms, for both bacilli and cocci have been described as the specific organisms. In many cases it is due to the bacillus prodigiosus.

The usual treatment recommended is shaving and subsequent bathing with 1 : 1,000 solution of corrosive sublimate. Although the concretions are described as very resistant to most solvents, I have had no difficulty in curing several cases of red lepothrix by simple bathing in 1 : 500 bichloride solution in alcohol.

II.—PIEDRA

ETYMOLOGY: (Spanish, *piedra*, a stone.)

Piedra is a disease of the hair occurring in Colombia, South America. It consists of nodular concretions on the hair produced by fungi. The concretions are pinhead size, and are multiple and hard, so that they rattle on combing. They occur on the scalps of native women, and occasionally on both the hair of the head and of the beard in men. It is due to a fungus showing mycelia and spores, which grows in a mucilagenous oil used for dressing the hair. Treatment consists in washing in antiseptics and such solvents as benzine or ether.

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[1] George T. Jackson, "Diseases of the Hair and Scalp," E. B. Treat, New York, 1894.

III.—CHIGNON

SYNONYMS: *Chignon Fungus, Beigel's Disease.*

This is an affection of both natural and false hair which consists of hard, nodular concretions due to the growth of masses of parasites strung along the hair shaft. The fungus is not definitely distinguished, and the condition is similar to, if not identical with, piedra.

IV.—TINEA NODOSA

SYNONYM: *Piedra nostras.*

Under this term Cheadle and Morris have described sheathlike, nodular incrustations developing on the hair of the beard and mustache from the growth of a fungus. The masses for the most part surround the hairs, which become brittle and break readily; but the fungi may penetrate the hair shaft, causing it to split. The concretions consist of mycelia and spores somewhat smaller than those of tinea tonsurans, and of a cement substance which the fungus secretes. Treatment consists in the use of antiseptics, but combined with this the hair must be shaved closely for some time.

Pseudo Concretions.—Pseudo concretions upon the hair shafts, near the bulb, are often seen in epilated hairs in various diseases. They are really portions of the internal root sheath which adhere to the hair shaft. They are found in various inflammatory processes in the hair follicles; may, indeed, be found where the hair is apparently normal. These masses of epithelium are called hair eaters, and are popularly regarded as the cause, and not the effect, of seborrhœic alopecia, in which they are very common.

V.—PLICA

SYNONYM: *Plica polonica.* ETYMOLOGY: Latin, *plica,* a fold or plait.

Plica, which was formerly regarded as a disease, is matting together of the hair by the accumulation of filth produced by the presence of innumerable parasites, usually pediculi. The hair may be matted together in the form of a feltlike mass on the head or in a dense cord. The condition was first described in Poles, but it may occur in any filthy subjects.

VI.—PLICA NEUROPATHICA

Le Page, Stelwagon, Ohmann-Dumesnil, and others have described cases of matting of the hair independent of dirt. They are apparently due to peculiar changes in shape or texture of the hairs which cause them to curl and twist so that they form tangled, hard lumps or elongated, ropelike masses. They are not due to cementing of the hairs together by extraneous substances, and are regarded as of neuropathic origin.

VII.—HYPERTRICHOSIS

Symptomatology.—Hypertrichosis is excessive growth of the hair. The term is purely relative. What is hypertrichosis in a woman would be regarded as deficient development of hair in a man. Hypertrichosis may consist in the growth of a few larger hairs than usual or in an abundance of down on the face of a woman, or it may consist of a growth of hair so excessive as to cover the surface with a growth like that of a hairy animal. It may consist either of an abundant growth of hairs of normal size or of a growth of hair of unusual length and coarseness. It is usually confined to parts normally hairy, but it may involve parts ordinarily supplied only with lanugo. In the most excessive cases of hypertrichosis an abnormal growth of hair will be present everywhere except on the palms and soles, where hair follicles are absent. Hypertrichosis, when once established, is usually permanent. In rare cases, however, it is produced by temporary nutritional disturbances, and when these disappear the hypertrichosis may also disappear.

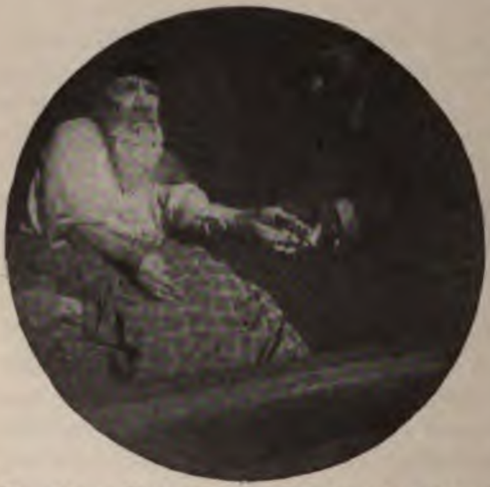

FIG. 335.—HYPERTRICHOSIS IN AN OLD WOMAN WHOSE SKIN SHOWED TEXTURAL CHANGES SUGGESTIVE OF MYXEDEMA. (Author's collection.)

Etiology.—The most important factor in hypertrichosis is family or racial peculiarities. There are characteristic racial differences in the amount of hairy development, as illustrated at one extreme by the almost hairless Australians and the negro, and at the other by the hairy Ainos of Japan. Families, also, may show an unusual degree of hairiness, and in such families the women may have a greater growth of hair upon he face than is usual. This individual tendency to hypertrichosis among women is most frequently seen in brunettes, especially in the form of a fine down. In both brunettes and blondes there may be an heredtary tendency to the growth of a greater or less number of coarse hairs upon the face.

In hypertrichosis the growth of the hair tends to become greater with age, and is particularly likely to develop in women about the climacteric. Independent of individual and family peculiarities, hypertrihosis in women may be a manifestation of some constitutional disturbnce. Menstrual and other disturbances of the sexual functions are at imes, but not frequently, the cause of the increased growth of hair. Very rarely it comes on after severe illness. Diseases of the suprarenal apsule, particularly carcinoma, may be accompanied by hypertrichosis. Occasionally an increased growth of hair is seen after injury to a member, such as a fracture, or a wound of a nerve.

At times hypertrichosis is produced by local irritation, as from hot dressings, stimulating local applications, or other severe irritants, like hus, but hypertrichosis of this origin is rare. There is a popular impression among women that the use of grease upon the face, especially aseline, may cause hypertrichosis. It is extremely doubtful if it ever does. In fact, although there are undoubted cases in which local stimuating applications have caused an increased growth of hair, most of the ases attributed to local causes must be viewed with skepticism. The desire of the patients to find some accidental cause for the blemish is very strong.

The extreme cases of hypertrichosis which are dermatological curiosities, like the Russian dog-faced man, are anomalies of development which show a strong hereditary tendency. Frequently the cases have occurred in family groups.

Treatment.—The radical treatment of hypertrichosis is difficult, nd, except for a comparatively small number of hairs, is likely to be unsatisfactory. Electrolysis thus far offers the only practical method or the destruction of hairs. The method can be used very successfully or individual hairs, but where the number of hairs to be removed is very large, there are practical difficulties which interfere with complete uccess. The method is very tedious, so that in some cases the treatment has to be very persistent, and there is always a minimum amount of carring. With care this can be made very slight, but without care and dexterity it may cause very considerable disfigurement of itself.

In the removal of hairs by electrolysis, a needle as fine as possible, preferably of platinum, is attached by means of a proper needle holder to the negative pole of the battery, while a sponge electrode is attached to the positive pole. A current of from two to five milliampères is sufficient. The needle should be inserted as nearly as possible into the hair follicle, and in order to do this it should be threaded into the follicle along the axis of the hair. The work is conveniently done by using an eyeglass of long focus or a hand lens which is held in the left hand between the fourth and fifth fingers while the surface of the skin is steadied by the thumb and middle finger. It is usually recommended to pass the current only after the needle is inserted, but in my experience it is better to make no effort to break the current either during insertion or removal of the needle. The current should be allowed to pass until bubbles of gas appear—only a few seconds—then, if the procedure has been successful, the hair can be pulled out without force. If the procedure has failed, it is better not to repeat it until the reaction has entirely subsided. Hairs closer to each other than one eighth of an inch should not be removed at the same sitting in order to avoid scarring. After the treatment the reaction is made less by bathing the parts in hot water several times during the day and the day following the electrolysis.

The treatment of hypertrichosis by x-rays was suggested by Freund, of Vienna, and is still recommended by him. Many others, including myself, have given the method a thorough trial. In my experience, practically satisfactory results can be gotten in about half of the cases; that is, the growth of hairs can be reduced to an amount which is not unsightly by repeated series of exposures which are safe; in the other fifty per cent, however, this cannot be done. The results are so uncertain, and the possibilities of harm are so great, that the treatment, in my opinion, should not be undertaken except in extreme cases, or with the full understanding that if the exposures are to be kept within the bounds of safety, improvement is altogether uncertain. The method of treatment consists esentially in repeated safe exposures, until the hair falls out—which may be the first evidence of x-ray effect in the most favorable cases—or until other evidence of reaction appears, when exposures should be immediately stopped. If the hairs fall out, they will begin to grow again in the course of six weeks to three months. Then the treatment has to be repeated; this repetition of treatment usually has to be carried out four or five times in the course of twelve to eighteen months. F. H. Montgomery, in a personal communication, tells me that he has seen the growth of hair stimulated by x-rays. In an experience with x-ray exposures extending over many thousands of exposures, I have never seen such an occurrence.

Occasionally patients demand some method of temporary relief from hypertrichosis aside from shaving or epilating the hairs. For this purpose, in most cases, the best thing that can be done is to keep the hairs bleached with hydrogen peroxide. To do this the hair should first be bathed with ammonia water, and then with a solution of hydrogen peroxide, or the hair can be thoroughly wet with equal parts of solution of hydrogen peroxide and ammonia water. Bulkley thinks that this constant bleaching with H_2O_2 retards the growth of the hair. Some cases of hypertrichosis are compelled to use depilatories. A type of these depilatories, and one of the best, is a mixture suggested by Duhring. This consists of barium sulphide, ʒij, with zinc oxide and starch, each ʒiij. The barium sulphide should be fresh. To use this mixture enough water is added to the powder to make a paste. This paste is thickly applied to the surface, and left for a few minutes until irritation begins. Upon removal the hairs, which the paste dissolves, are removed with it. The slight redness, if the paste is not left on too long, is relieved by a dusting powder or bland ointment. The depilation has to be repeated once in a week or two, and, like shaving, makes the hairs coarser.

VIII.—FRAGILITAS CRINIUM

Fragility of the hair manifests itself either in brittleness, so that the hairs break off, or in tendency to split. In the breaking type of the condition the hairs break off from handling. In the splitting type the tendency to split is usually at the tip of the hairs, and this type of fragility is a common condition in long hair. Usually the splitting is limited to the tip. Occasionally the hair will split in its entire length. In some other cases a splitting up of the hair in the shaft without involvement of the tip occurs, and even the splitting has been seen to begin in the follicle. In these last cases there is usually folliculitis, but not in the others. The condition may be confined to a few hairs or occur in the majority of them.

Fragility of the hair may be produced by parasites, especially ringworm and favus, but these forms are not included under the term fragilitas crinium. The condition is apparently a nutritional disturbance of trophic origin, concerning which nothing is known. The hair roots may be normal, or in some cases show atrophy, as noted by Duhring.

Treatment.—The brittleness and splitting is in part overcome by oiling the hair—which is usually abnormally dry. Beyond this nothing can be done for it. Where the splitting occurs at the tips, it can be prevented from extending by clipping off the split ends. Nothing is gained by the barber's common procedure of singeing.

IX.—TRICHORRHEXIS NODOSA[1]

SYNONYMS: *Nodositas crinium, Trichoclasia, Clastothrix.*

Trichorrhexis nodosa is a nutritional disturbance of the hair in which minute swellings develop along the hair shaft and incomplete fractures of the hairs occur at these points. The nodes appear as minute whitish swellings, of which there may be half a dozen or more along the hair shaft. The hair breaks incompletely at these points, the cortex splitting into filaments while the medulla remains intact. It is really a sort of green-stick fracture, whose appearance has aptly been compared to that of two shaving brushes pressed end to end. The affection attacks especially the beard and mustache; less frequently the eyebrows, and the hairs of the scalp, axillæ, pubes and genitals.

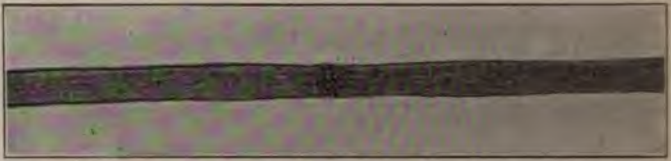

FIG. 336.—TRICHORRHEXIS NODOSA. (Author's collection.)

The cause of the condition is not definitely determined. Various organisms have been described by Raymond, Blachko, Hodara, Spiegler, and others, but the pathogenic character of none of them has been demonstrated. In some cases the disease has been apparently spread by infection; Hodara, indeed, claims to have succeeded in producing it by inoculation. Crocker has seen it in husband and wife, and Ravenel of Charleston, who had it in the mustache, found the same condition in his shaving brush and toothbrush. It is possible that the disease may be produced by general nutritional disturbances, and also by parasites, the result in either event being fragility of the hairs, which is followed by incomplete fracture upon bending.

Treatment is not satisfactory. Shaving may or may not do good, and the same is true of the local use of antiseptics.

TRICHONODOSIS

Under this name Galewsky has recently described from two cases a peculiar knotting and breaking of the hairs, resulting in partial alopeia and interference with the hairs' growth in length.[1] Microscopically he hairs were found to be oval or flat and often twisted on their long xes. This peculiarity in the forms of the hair causes a curling, and s a result of this twisting and curling of the hair shafts, there form ops, spirals and simple and looped knots. At these knots the hair reaks off, leaving atrophic or frayed ends. Near the ends some of the airs show nodular thickening. Of course this breaking of the hair at he knots accounts for its failure to attain the usual length. In Gaewsky's first case there was an apparent premature alopecia. The hair as thin, somewhat atrophic, and easily pulled out. The condition exsted in the hairs of the scalp, the beard, the trunk, the genitals and the highs.

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[1] Raymond, *Annales*, 1891, p. 568.—Ravenel, *Medical News*, October, 1892.—Hodara, *Monats.*, vol. xix, 1894, p. 173.—Spiegler, *Wiener med. Blätter*, 1895, p. 399.—Essen, *Archiv*, 1895, vol. xxxiii, p. 415.—Barlow, *Münch. med. Wochenschr.*, 1896, p. 651.—Jackson, *Jour. Cutan. Dis.*, 1903, p. 473.—Heidingsfeld, *Jour. Cutan. Dis.*, June, 1905, p. 246 (literature and bibliography).

MacLeod has reported another case in which the changes in the hairs ere the same as in Galewsky's cases. In this case the condition was not haracterized by distinct alopecia, but the patient sought advice because er scalp hair failed to grow to the usual length and had a moplike tousled appearance. None of these patients had subjective symptoms.

The etiology of the condition is not determined. Galewsky's first ase was a healthy young man; his second an anæmic, weak girl. In acLeod's patient, a Singhalese girl, the condition first attracted attenion after her hair had been cut during a severe attack of pneumonia. n Galewsky's first case the patient's father had apparently had the ame condition, but there was no evidence of heredity in the other ases. In all of these cases the condition had existed for years and all ere unaffected by treatment. Veiel of Canstatt, Schiff of Vienna, Juleld of Berlin, Schmitt of Dresden, Bohm of Chemnitz have reported o Galewsky similar cases which they have observed and Galewsky thinks he condition is probably not very rare.

Saalfeld[2] has suggested that Michelson, Bulkley, Jackson, and himelf have previously described the same condition in the pubic hairs, in ases in which the condition was accompanied by severe itching; and e believes the condition is largely a mechanical one produced by angling the hairs in scratching. The absence of itching in the afection as observed by Galewsky and MacLeod,[3] its wide distribution over he body, and the peculiarities in the forms of the hair, would seem o indicate that the condition referred to by Saalfeld is not the ame.

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[1] Galewsky, *Archiv*, 1906, B. 81, p. 195.

[2] Saalfeld, *Archiv*, 1906, B. 82, p. 245.

[3] MacLeod, *Brit. Jour. Derm.*, 1907, p. 40.

X.—MONILITHRIX[1]

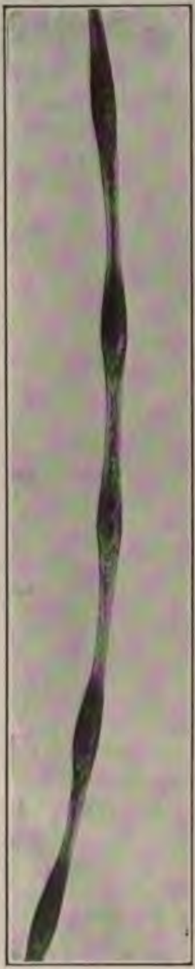

FIG. 337.—MONILIFORM HAIR. × 45. (Author's collection.)

SYNONYMS: *Moniliform Hair, Beaded Hair.*

Monilithrix is a condition in which the hair shafts show fusiform swellings separated by portions which are constricted. The fusiform swellings represent the normal hair shaft and are pigmented, while the constrictions are narrower than the normal shaft, and are of lighter color. The hairs accordingly have a ringed appearance, and are weakest in the constrictions. The moniliform structure extends down the root of the hair. The condition usually affects all of the hairs, but may, as in Gilchrist's case, be limited to certain areas. The affection is frequently associated with lichen pilaris.

The affection is in nearly all cases congenital, and is present from infancy. It may occur in family groups, and shows a marked hereditary tendency; Sabouraud has traced it through five generations in one group of cases. A few cases, reported by Gilchrist, Morrow, Walter Smith, and Unna, have been observed that appeared during adolescence or later.

The condition is probably due, as suggested by Virchow and Kaposi, to periodic aplasia of the hair papillæ. During these periods the parts of the hair shafts which are produced are not fully developed, while in the normal intervals the parts of the hair shafts which are produced are of normal diameter. Search for organisms in the affection has been without results.

Treatment is without benefit. In one area in Gilchrist's case the disease, after several years, disappeared spontaneously.

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[1] Beatty and Scott, *Brit. Jour. Derm.*, 1892, p. 171 (review and bibliography).—Gilchrist, *Jour. Cutan. Dis.*, 1898, p. 157 (review and bibliography).—Morrow, *Jour. Cutan. Dis.*, 1899, p. 41.—Ruggles, *Jour. Cutan. Dis.*, 1900, p. 500.

COLOR CHANGES IN THE HAIR

XI.—CANITIES

SYNONYM: *Grayness.*

Symptomatology.—Canities (Latin, *Canus*, gray) may be congenital or acquired. Congenital canities may be complete, corresponding to complete albinism, or it may be partial, consisting of circumscribed areas of white hair corresponding to partial albinism. In congenital canities the hair is white in affected areas. In acquired canities the hair varies in color through all shades of gray to white. The condition usually develops gradually by the blanching of individual hairs, and upon the relative proportion of white hairs depends the degree of grayness. Usually the individual blanched hairs are devoid of pigment throughout their entire length. Occasionally the lower part of the hairs is pigmented while the distal part is white. In very rare cases the hair may be pigmented at the tip and white in the lower part.

Occasionally canities is unsymmetrical in distribution. This is seen in rare instances where grayness takes place in the area of distribution of a diseased or injured nerve. It is not infrequently seen in the circumscribed white patches from one to two or three inches in diameter, most frequently at the front of the scalp, which are the result of partial albinism. Similar patches may be due to leukoderma, or to new hairs growing in patches of alopecia areata. Grayness resulting from nerve lesions or leukoderma or alopecia areata may be temporary. Other forms of canities when once established are permanent, although there are a few cases in literature in which the hair has regained its color. There are some well-authenticated cases of sudden blanching of the hair, nearly always after severe nervous shock. Such careful observers as Raymond, Pincus, Landois, and Gowers have recorded cases in which the hair turned gray in from five hours to three days. Cases in which the hair has turned gray in the course of a few weeks are less uncommon.

Ringed Hairs.—A few curious cases have been recorded of ringed hairs, in which rings of white alternate with pigmented rings in the shafts of the hairs. The hairs show microscopically successive parts of the hair shaft which are alternately pigmented and unpigmented.

Etiology and Pathology.—Acquired grayness is a senile change. Its development is largely controlled by heredity, but it may at times be hastened by severe illness or other general disturbance. The influence of the nervous system in the production of grayness is often very definite, as shown especially by grayness occurring in areas of nerve injury or disease or as a result of grief, anxiety, or other nervous strains.

Whiteness is due, as a rule, to the lack of pigment in the hair shaft. This probably results from failure of its production by the pigment-forming cells of the papilla, but it possibly results, as Ehrmann suggests, not so much from failure of production of pigment as from lack of its transmission into the hair through the absence of pigment-bearing cells. Landois has shown that whiteness of the hair may be produced by the formation from some cause of air bubbles in the corticle layers, and this phenomenon is probably the explanation of the grayness which appears suddenly.

Treatment for grayness is without avail.

XII.—DISCOLORATIONS OF THE HAIR[1]

Rare cases of change of color of the hair are observed. Prentiss has reported a case in which the hair turned black from hypodermic injections of pilocarpine continued for two months; the darkening of the hair began within twelve days after beginning with pilocarpine, and increased for six months. Alibert and Beigel have recorded cases in which light hair, after falling out as a result of severe illness, grew in black. Alibert also records a case where brown hair, lost in an illness, returned as red. Several cases of change to reddish color have been observed, usually in epileptics or in lunatics, or in brain disease, the change in some cases being permanent, in others the color varying. I have seen light hair become jet black in Addison's disease; darkening of the hair is not, I believe, a rare occurrence in cases of extreme pigmentation in that disease.

XIII.—ALOPECIA

SYNONYMS: *Calvities, Acomia.*

Alopecia is baldness. It may be partial or complete in the areas involved, and it may be circumscribed or universal. It may be congenital or acquired. In all forms of gradually developing alopecia, baldness results not from the sudden falling out of individual normal-sized hairs, but from the fact that as the hairs fall out finer hairs regrow in their place, and this gradual decrease in size of the hairs continues until they finally cease to appear. In alopecias developing rapidly, as in those produced by infectious diseases, the baldness is the result of the sudden shedding of hairs of normal size. When these regrow they may be quite as large and vigorous as formerly.

Congenital alopecia is baldness due to lack of normal development of hair follicles. It is usually partial, but it may be complete. As a rule, it is associated with other defects, especially of the nails and teeth, and there may be lack of development of the other appendages of the skin. According to Hill, there are some Australian tribes who are normally hairless.

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[1] Jackson, *Jour. Cutan. Dis.*, vol. ii, p. 173.

Acquired alopecia may be divided into:

(1) Symptomatic alopecia.
(2) Senile alopecia.
(3) Idiopathic premature alopecia.
(4) Alopecia areata.

Symptomatic alopecia may be due to constitutional or local causes.

ALOPECIAS OF CONSTITUTIONAL ORIGIN are seen in the course of or after infectious diseases, like the various specific fevers. The alopecia which occurs in secondary syphilis is of the same character. In these alopecias the nutrition of the hair is damaged by toxæmias and the hair rapidly falls out, though usually only partly. After the toxæmia disappears regrowth rapidly sets in, and complete, or almost complete, restoration of the hair occurs.

A unique form of alopecia of toxic origin is sometimes seen from the administration of thallium acetate for excessive sweating in tuberculosis. Many cases of alopecia of this origin have been observed. The alopecia disappears after the drug is discontinued.

Alopecia of constitutional origin sometimes occurs from the general failure of nutrition in cachexias from various causes, like tuberculosis, diabetes mellitus, myxœdema, and leprosy. In these conditions the alopecia is an expression of profound disturbances of nutrition, and is usually permanent; it is, however, always only partial.

In all these forms the alopecia, as a rule, entirely disappears upon the restoration of the general health. In alopecias due to infectious diseases the regrowth of hair is usually normal; sometimes it is less, but occasionally it is even more abundant than before.

Alopecias dependent upon conditions of general lowered nutrition may or may not improve if the general health is restored. These cachectic alopecias in many cases are of the same character as the so-called idiopathic alopecias.

The treatment of these alopecias of constitutional origin is largely internal. In syphilitic alopecia, for example, the administration of mercury rapidly controls it. Locally, the cases are treated by the methods of stimulating the scalp which are considered under the treatment of alopecia in general. Nothing is gained in alopecias from acute infectious diseases by cutting the hair short, as is so commonly done.

ALOPECIA FROM LOCAL CAUSES may be due to various local destructive processes. It may result from all sorts of pathological conditions which, either through ulceration or through the production of scar tissue without ulceration, cause destruction of the hair follicles. Among the conditions which produce bald scars of this sort are the following:

Kerion (tinea).
Favus.
Ulcerating syphilides, especially gummata and large tubercular syphilides.
Folliculitis decalvans.
Confluent smallpox, where the scars are deep and large.
Lupus erythematosus.
Morphea.

Alopecia occurring in scars is irregular, and is permanent.

Alopecia of local origin may also result from persistent inflammatory diseases of the scalp, like psoriasis, eczema, and seborrhœic dermatitis, which gradually produce sclerosis and destruction of the follicles. It is surprising, however, how rarely psoriasis, although it habitually affects the scalp, and eczema cause baldness, but seborrhœic eczema usually in the end results in baldness.

In seborrhœic alopecia there is gradual thinning of the hair, beginning on the temples and slowly invading the vertex and front of the scalp. It is accompanied by more or less dandruff, and in its early course by oiliness; later the hair becomes dry, lusterless, and grows less and less vigorously until it entirely disappears in the invaded areas.

Symptomatic alopecia may be produced by parasitic diseases which directly destroy the hair. This is best illustrated in ringworm, where temporary, and usually partial, alopecia occurs in the affected areas.

Alopecia from local destruction of the hair without disease sometimes results from mechanical causes. It may be produced by the friction of hats or headbands or from damage to the hair in dressing it, as from excessive use of curling irons. Occasionally it may result from an unconscious habit of pulling out the hair. I have seen this illustrated in the case of an old woman with a round patch of alopecia on the front of the scalp, which was ultimately discovered to be due to the habit of pulling out the hair at this point while she was reading.

The prognosis and treatment of most of these forms are evident from their etiology, and do not need further consideration. The only common form of baldness, as the term is ordinarily understood, included in this group is seborrhœic alopecia.

Senile alopecia—the baldness of old age—begins at the vertex and progresses as a gradually spreading, round area, which finally involves the crown and front of the scalp, but nearly always leaves a fringe at the sides and back of the scalp. It is one of the senile changes in the skin, and is a manifestation of atrophy of the skin in general. The reason usually offered for its appearing at the vertex is that the scalp is thinnest there, and is most promptly affected by atrophic changes.

Idiopathic premature alopecia is in some cases identical with senile alopecia, simply developing before the usual senile period and before other senile changes are manifest. This form of idiopathic alopecia, like senile alopecia, usually begins on the vertex. It may be seen as early as twenty-five. Most of the so-called premature idiopathic alopecias, however, are symptomatic alopecias due to seborrhœic dermatitis,[1] probably the commonest cause of baldness.

Etiology of Senile and Premature Alopecia.—As already stated, senile alopecia, idiopathic premature alopecia, and alopecia from seborrhœic dermatitis are the usual forms of alopecia. It is impossible to separate seborrhœic alopecia entirely from other forms of premature alopecia, so they must to a certain extent be considered together.

Seborrhœic alopecia is produced by the causes that produce seborrhœic eczema, which have been considered under that disease. It is also possible that many of the factors which are supposed to contribute to the production of premature idiopathic alopecia are as well contributory causes in the production of seborrhœic dermatitis and seborrhœic alopecia.

Alopecia is perhaps commoner now than formerly, and it is very much more frequent in men than in women. The reasons for these two facts are uncertain. If alopecia is more common now than formerly, it is due doubtless to changes in methods of living and in the environments of civilization; but what the unfavorable factors are in these changes we do not know. The reasons also for the greater frequency of alopecia in men than in women can only be surmised.

One of the causes which is assigned for the production of baldness in men is the wearing of stiff hats, which interfere with the circulation of the scalp by compressing the temporal arteries (Jamieson) and prevent free circulation of air and exposure to light. It is doubtless true that if men never wore hats, the hair of the head would rise to the demand for the exercise of its protective function and grow more abundantly, and to that extent the wearing of hats is probably a factor in the cause of baldness. Now, however, that men are accustomed to the wearing of head covering, I believe that the fashion of going without hats, especially during the summer time, which is in great vogue at present, may be an exciting factor in causing baldness. It is entirely possible that the effect upon the scalp produced by light may influence the hair follicles unfavorably, in the same way that x-rays act, although to a less degree. It is at least my experience that numerous cases now appear for treatment for falling of the hair after sojourns at the seashore or in the country, where the individuals have intentionally gone without hats in order to stimulate a flagging growth of hair. That brain work is a cause of baldness and that baldness is an affliction of the intellectual is a favorite and grateful theory. Baldness is perhaps considerably more common in individuals of the class who lead an indoor life, but this class is physically less vigorous in many ways than the manual-laboring class, and it is extremely doubtful, in my opinion, if the baldness of the intellectual laborers is due to mental work rather than to many other of their habits which are different from those of the manual-laboring classes. The premature baldness of the sedentary class is, in my opinion, probably due to the various factors which combine to make sedentary life less vigorous than a life demanding manual labor.

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[1] Elliot, *New York Med. Jour.*, October 26, 1895.

The influence of proper attention to the scalp is perhaps the most important factor in the production of baldness. In the first place, a moderate seborrhœic dermatitis is usually uncared for until baldness is well advanced. The scalp is not kept free from dandruff, the hair and scalp are brushed no more than is necessary to keep the hair in place, and frequently the hair and scalp are wet daily, either as an aid in arranging or in an unintelligent effort to keep the head clean. In the better care which women take of their hair, in the difference in the dressing of the hair, and in the hats worn probably lie the reasons for the lesser frequency of baldness in women than in men. As a rule, they never wet the hair with water without thoroughly drying it. The scalp is kept clean by brushing and an occasional shampoo, and the hair is allowed more completely to fulfill its function of protective covering for the scalp, for women habitually wear hats which are merely ornaments and furnish no protection to the scalp.

Treatment.—The treatment of baldness is practically exclusively local. General tonics and specific remedies directed to the scalp are, in my opinion, utterly useless. General tonics, where indicated, may do the patients good, but I have never had any reason to believe that they made any changes in the growth of the hair except in symptomatic baldness arising from constitutional diseases.

Local measures, in the first place, are directed to keeping the scalp clean and in overcoming seborrhœic eczema. There is no objection to washing the scalp as often as necessary for cleanliness—that is, from once in several days to once in two or three weeks—but after this the scalp should be thoroughly dried. Some attention daily to brushing the hair is, I believe, of service in preventing alopecia, both by cleaning the scalp and by the stimulation which it affords. It certainly keeps the hair glossy, and in better condition cosmetically.

Especial attention should be given to the treatment of dandruff and to more exaggerated degrees of seborrhœic eczema. The treatment of both of these has been taken up under the head of seborrhœic dermatitis.

Aside from the treatment of seborrhœic dermatitis and the very exceptional inflammatory diseases of the scalp which produce baldness, the treatment of baldness consists chiefly in the use of stimulating measures. These do not increase the growth of the hair, but they perhaps, to a certain extent, check the falling of the hair. Stimulating applications are used both in the form of lotions ("hair tonics") and of ointments or pomades. Ointments or pomades may be indicated for seborrhœic dermatitis or other inflammatory diseases of the scalp, but where they are to be used as stimulating applications they are less desirable than lotions. Lotions may be made either with water, witch-hazel, alcohol, bay rum, or cologne. The aqueous solutions are less desirable than solutions containing at least fifty per cent of alcohol, for the reason that they do not dry readily, and have to a certain extent the same objections which obtain against the daily wetting of the scalp. Alcohol solutions are particularly desirable where the hair is very oily. If they make the hair very dry, or if the hair is already dry, this can be overcome by adding from ½ to 2 drams of castor oil to the pint of alcohol. Glycerine in the same proportions is used, but is less desirable. In place of using a small amount of oil in the tonic, the dryness can be overcome by rubbing into the scalp a small quantity of vaseline or cold cream. Many stimulating remedies are recommended for alopecia, but all that can be done with any of them can be done with tincture of cantharides, tincture of capsicum, corrosive sublimate, and formalin. Suitable prescriptions of these are as follows:

Tincture of cantharides, Tincture of capsicum, } āā	ʒiv–℥i;
Alcohol	O.i.

Formalin	ʒi–iv;
Alcohol	O.i.

Corrosive sublimate	gr. viiss–xv;
Alcohol	O.i.

Any of these prescriptions, of course, can be made up with spirits of bay rum instead of alcohol, or, if alcohol is used, a few drams of cologne can be added for those who like that sort of thing. These preparations should be applied to the scalp thoroughly once a day, and their effect can be increased by rubbing. When any of them produces perceptible irritation, its use should be stopped until this disappears, and as a rule the strength of the application used subsequently should be reduced to the point where it does not cause irritation. The daily, or at least frequent, use of such tonics as these as part of the toilet is of considerable service in checking the course of alopecia. This, perhaps in most cases, is due rather to the effect in keeping the scalp clean and especially in controlling seborrhœic eczema, than to their stimulating effects *per se.*

For the purpose of stimulating the scalp and to prevent its atrophy, the best measure is probably daily massage by the patient himself. This has the strong indorsement of Jackson, and consists essentially in pinching and vigorous rubbing of the scalp with the finger tips until there is produced a sense of warmth. Another stimulating measure which may be of service is the application to the scalp two or three times per week of a faradic current. This is applied either through a metallic brush or a comb, and the scalp is gone over with this for five or ten minutes, or until some hyperæmia is produced.

XIV.—ALOPECIA AREATA[1]

SYNONYMS: *Porrigo decalvans, Area celsi, Alopecia circumscripta.*

Alopecia areata is a disease of the hair which is characterized by sharply circumscribed patches of total baldness.

Symptomatology.—The baldness in alopecia areata occurs in patches of various size and shape. Ordinarily the patches are rounding, and from one to two or three inches in diameter. They may, however, be of quite irregular shape, and occasionally are band form. The skin of the affected areas is entirely devoid of hair, is usually quite white, and is perhaps slightly depressed, or has an atrophic feel. Occasionally the skin is pinkish, and very rarely is even slightly elevated. Where the patches are stationary the line of demarcation of the bald patches is usually sharp, the hair beyond the border of the patch being normal and of ordinary vigor. In patches, however, which are spreading, the hairs at the borders may be loose and fragile. The presence of loose, broken hairs is also found at the border of some of the cases which presumably are of parasitic origin. The distribution of alopecia areata is altogether irregular. There is, as a rule, no symmetry in the distribution of the patches, and no evidence of anatomical or physiological factors to account for the arrangement. Occasionally they may have the distribution of a definite nerve area, or occur at the site of previous injury. The disease is usually confined to the scalp. It may, however, appear upon the hairy parts of the face, and in occasional cases upon other parts of the body. Frequently the entire manifestation of the condition is limited to one or two coin-sized patches. As a rule, there are several patches of different size on the scalp. In some cases the patches are so large and numerous on the scalp that only irregular areas covered with hair are left. These confluent areas of baldness, however, are not usual, the patches tending to remain discrete.

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[1] Joseph, *Monats.*, 1886, p. 483.—Pontoppidan, *Monats.*, 1889, vol. viii, p. 51.—Sabouraud, *Annales de l'Institut Pasteur*, 1897, p. 134.—Bender, *Dermatolog. Centralbl.*, October, 1898.—Bowen, *Jour. Cutan. Dis.*, 1899, p. 399.—Heidingsfeld, *Cincinnati Lancet-Clinic*, March 3, 1900.—Robinson, Morrow's "System," vol. iii, p. 862.

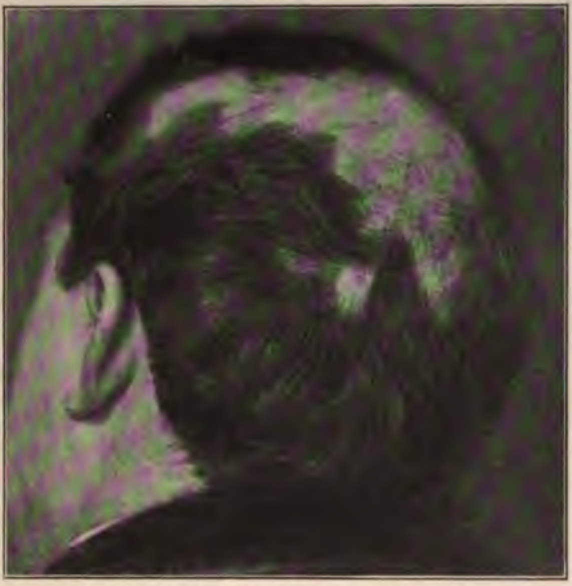

FIG. 338.—ALOPECIA AREATA OF ORDINARY TYPE, BUT PATCH SOMEWHAT LARGER THAN USUAL. (Author's collection.)

Universal Alopecia.—Alopecia areata may become universal by the rapid development over the body of patches which become so abundant as to involve the entire surface. In some of these cases the alopecia is absolutely universal, involving eyelashes, bristle hairs about the orifices, as well as the lanugo and coarse hair. Another form of universal alopecia occurs which is probably of the same character as the universal alopecia which begins in patches, but in this form rapid shedding of the hair occurs diffusely over the body.

The onset of alopecia areata is sudden. The first evidence of it is the appearance of a well-developed bald patch. The hair from such a patch may fall out suddenly and the patch remain of its first size. In other cases the patches spread peripherally. In some cases the disease may not extend beyond the formation of one patch, but as a rule subsequent patches appear irregularly. The course of the affection is altogether capricious. Hair may grow rapidly upon the patches without discoverable reason, and after a certain amount of growth fall out and grow again. The young hair which grows in the patches is usually light color and readily falls out, but when a vigorous growth of hair develops it regains its normal color and may be permanent. In the usual case the disease continues for from one to several years. Occasionally the affection disappears in a few months. On the other hand, in the extreme cases the disease may persist for years, disappearing, however, in most cases ultimately. The universal cases are the most persistent; those beginning in patches and in the young are apt to recover in time but many years may elapse before this happens; those which begin with diffuse rapid falling of the hair are apt to be permanent. There are no subjective symptoms of the disease. The patches are, if anything, slightly anæsthetic. The onset of the trouble is usually without subjective symptoms, but in very rare cases there is a history of headache, or malaise, or of some other constitutional disturbance, and at the site of the patches there may be a preceding pruritus or formication.

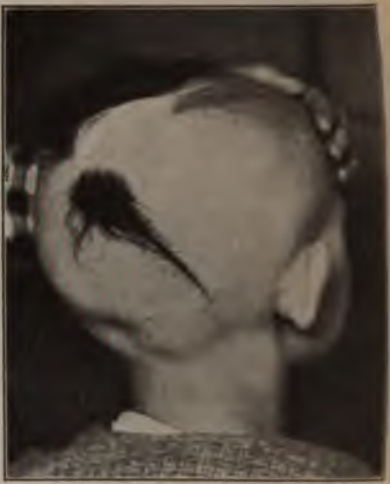

FIG. 339.—ALOPECIA AREATA, UNUSUALLY EXTENSIVE. The alopecia had previously been complete and universal. (Author's collection.)

Etiology and Pathology.—The etiology of alopecia areata is altogether obscure. It is more frequent in my experience in men, and is no respecter of social classes. It is not at all uncommon in children, although it is rare before five years of age. It is most frequently seen in early or middle adult life.

In many cases no causative factors can be discovered; the patients are often vigorous, healthy children, of well-balanced nervous systems, and with no suspicion of contagion to account for the disease. In another group of cases, however, there is strong ground for suspecting a nervous origin. Many cases have been reported in which the disease seemed directly traceable to nervous shock, anxiety, and worry. In rare cases in which the patches follow a nerve area the disease is apparently the result of traumatic neuritis. In one group of cases Stelwagon has obtained a history of heredity, the affection having appeared in three generations.

There is still another group of cases in which the evidence of parasitic origin is strong. Thus many epidemics in various localities and institutions have been observed, especially in France. One such epidemic has come under my observation in a town of several thousand inhabitants. Bowen has described two remarkable epidemics which occurred in an institution for girls in Boston. In the first epidemic sixty-three of sixty-nine inmates of the institution showed the affection, and the second epidemic began with the readmission into the institution of the same girl from whom the first epidemic started. But these epidemics are very uncommon, and when the disease is contagious the contagion is very weak.

From consideration of the different groups of cases there is little doubt that two forms of alopecia areata exist—one a trophoneurotic disease, the other a parasitic. In favor of the trophoneurotic origin of some of the cases are the experiments of Joseph and others, in which the excision of the second cervical ganglion was followed by the appearance of bald patches in the areas of distribution of the second cervical and the auricular and occipital nerves. Areas of baldness have also been observed by Pontoppidan and Bender after operations involving the nerves of the neck. Various organisms have been described as pathogenic of the disease, beginning with Gruby in 1843 and coming down to Sabouraud. Sabouraud regards the disease as due to his ubiquitous microbacillus, and thinks it is an acute seborrhœa oleosa. He has not established his position, and his views are not widely accepted.

Microscopically the skin shows atrophy of the pilo-sebaceous follicles. The studies of Robinson, Giovannini, Unna, and others have shown that the disease is inflammatory. Robinson, who has examined a large number of sections, finds that it is an inflammatory process showing as a perivascular infiltration in the body of the corium, and to a much less extent in the papillary layer. The roots of the hairs which fall out of the patches show the changes of simple atrophy.

Diagnosis.—The characteristic feature of alopecia areata is the presence of bald patches which are smooth, usually white and slightly atrophic, and have appeared suddenly without preceding destructive disease of the skin. They may be confused with bald patches of lupus erythematosus or with other bald scars on the scalp, but a little consideration of the texture of the bald patches in these diseases, and of their history, is sufficient to differentiate them from alopecia areata. The condition is most likely to be confused with ringworm of the scalp. In this the patches are not entirely bald, and are not smooth except in the rarest of cases. They are more or less scaly, show numerous broken hairs, and the fungus can readily be demonstrated.

Prognosis and Treatment.—The prognosis as regards recovery alopecia areata is less favorable the older the patient and the more e tensive the baldness. In young patients, except in the universal cas

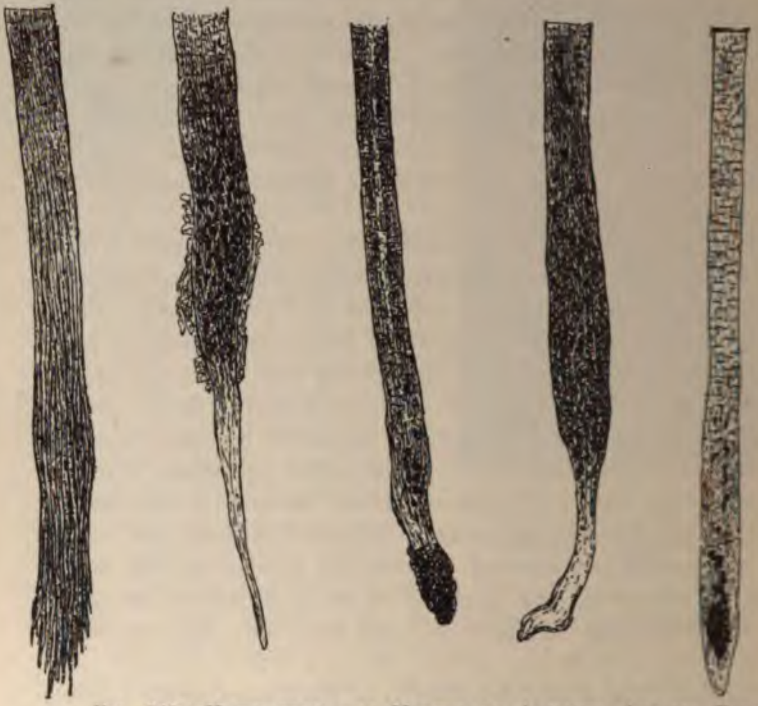

FIG. 340.—HAIRS FROM THE MARGIN OF ALOPECIA AREATA PATCHES. (Robinson, Morrow's system.)

recovery ultimately takes place. The same is true of most adult ca under forty-five, except where the baldness is very extensive. The v extensive cases in the elderly may or may not get well. The con of the universal cases has already been referred to. A favorable sign recovery is the growth of down over the affected areas. This may f out again and again, but usually ends in a regrowth of normal ha In patients who are below normal health, tonics like cod-liver oil, ir quinine, and strychnine are regarded as useful. In the cases of nerv origin there is reason to believe that the administration of arsenic moderate doses may be distinctly beneficial. Pilocarpine, also, is believ to be useful, and is recommended in doses of $\frac{1}{30}$ to $\frac{1}{16}$ grains sub taneously.

The chief reliance, however, must be placed upon local treatme As a matter of precaution, in cases where there is any possibility contagion, measures should be taken to prevent spread of the disea

Each patient should be careful to confine himself to his own toilet articles and to his own headwear. As a rule, the patient should have a stimulating antiseptic application for use over the entire scalp, with the hope that it may prevent the spread of the disease by autoinfection. For this purpose nothing is better than a solution of corrosive sublimate, 7½ to 15 grains, or of formalin, 1 to 3 drams to the pint of alcohol. The entire scalp should be wet with this solution at least once in three or four days.

The remedies which are used upon the patches are all stimulating antiseptics. The list includes almost all of the irritating agents which are used in dermatology, among them tar, beta-naphthol, corrosive sublimate, chrysarobin, tincture of capsicum, tincture of cantharides, and carbolic acid. Apparently it does not make much difference what application is used, provided it is strong enough to produce stimulation of the parts. In my experience, no application is better than ninety-five-per-cent carbolic acid, as recommended by Bulkley, or, upon irritable parts, equal parts carbolic acid and glycerine. On the scalp the patches are less irritable than those upon other parts, and as a rule they bear strong applications well. Ninety-five-per-cent carbolic acid can be applied to them without danger of sloughing if it is not repeated until its effect has disappeared. Upon other parts than the scalp carbolic acid may be used with equal parts glycerine or alcohol. Another good mixture, where undue irritation is to be feared, is equal parts carbolic acid, tincture of iodine, and chloral hydrate, which is to be painted on the patches at intervals of a few days. Next to these applications in preference are perhaps chrysarobin, from 10 to 60 grains dissolved in 1 ounce of chloroform, and corrosive sublimate, ½ to 2 grains to the ounce of water or alcohol. The chrysarobin solution is painted over the surface and allowed to dry, and is then covered with a layer of flexible collodion. The application used should be applied for about one quarter inch beyond the border of the bald patch. Applications of whose effect one is sure can be applied to all the bald patches at one time. Pure carbolic acid should not be used indiscriminately or extensively at first, but should be applied to limited areas until it is determined that the application is safe. In the application of any of these irritating remedies their use should be stopped when dermatitis is produced, and not renewed until it has subsided. The patches can also be stimulated by the application of a galvanic current or of a high-frequency or static current, and these are all recommended. In my opinion, they offer no advantages over the less troublesome chemical stimulants. The application of ultraviolet rays has been used with benefit in Copenhagen and elsewhere, but it is often disappointing. Reports of cures also from the use of x-rays have been made, but no reliance can be placed upon their effect.

FOLLICULITIS OF THE

XV.—FOLLICULITIS

Synonyms: *Alopecia follicularis* (Hyde. an
(Quinquad), *Alopécie cicatricielle innominée* (Be
Roberts), *Lupoid sycosis* (Milton, Brocq), *Uler*
pelade (Brocq), *Alopecia circumscripta seu orbicul*
(Crocker).

Under these various names have been
the essential characteristic is folliculitis
by the formation of punctate scarring an
that in time the process, which is always
Two types of the affection may be con
which occurs upon the scalp, and lupoid
bearded part of the face. Most of these
common pus organisms, and are forms o
sycosis on the face is probably in some c
analogous to acnitis. But the pathology
is not well enough determined to permit

Symptomatology.—The essential lesi
groups of small inflammatory papules o
hair follicles and are pierced by hairs.
undergo slow involution with the prod
lesions are usually grouped in adjoining
be scattered. The condition is persiste
border, so that gradually it produces sli
the centers of which are made up of w
tissue, while at the border the active lesi
destroys all of the follicles in the invade
may be left growing in the patches. T
but may appear in the pubic and axilla
part of the face. The course of the c
Its spread is very slow, but persistent.
cence, and the disease may disappear spo
progressive, and while it heals in certai
other points.

The French describe another variety
the term *pseudo-pelade* (pseudo-alopecia
course of the affection is the same as in
except that the inflammatory manifestatio
faction of the hair follicles. The patches
those of alopecia areata, except for the
border.

Folliculitis decalvans usually develops between thirty and forty, and most frequently in men of the laboring classes. The cause of the disease is not known, although it is almost certainly due to infection, probably with common pus organisms. Anatomically the condition is a perifollicular inflammatory process, usually of low grade. The condition has to be distinguished from alopecia areata and lupus erythematosus of the scalp. The evidence of slowly spreading folliculitis and the slowly progressive course of the patches are sufficient to distinguish it from alopecia areata. In lupus erythematosus the erythema is more diffuse, the patches, particularly at the borders, are indurated, and the scarring is greater, and not of the punctate character of folliculitis decalvans.

Treatment.—Treatment is by the use of local antiseptics, and is similar to that for sycosis and tinea tonsurans. The hairs at the border should be epilated, and an antiseptic ointment of sulphur, ammoniated mercury, resorcin, salicylic acid, or beta-naphthol applied daily. The patches may be irritable, and the application at first should not be strong. The scalp should be washed at intervals of a few days with an antiseptic application, like 1:1,000 corrosive sublimate, in alcohol or alcohol and water. X-rays should be tried.

XVI.—LUPOID SYCOSIS (Milton, Brocq); ULERYTHEMA SYCOSIFORME (Unna)

This occurs upon the bearded part of the face as a chronic papular or pustular folliculitis which spreads peripherally. The hair follicles are destroyed by the folliculitis, punctate atrophic scars are formed, and baldness in the involved areas occurs. The progress of the disease is extremely slow, but in the end it may produce marked scarring which may be either atrophic or keloidal. The disease may present a close resemblance to sycosis from infection with pus organisms, but its much slower course, the marked scarring and baldness which it produces, and the more indolent character of the lesions are sufficient to distinguish it from sycosis. The etiology is uncertain. Hyde and Montgomery have found tubercle bacilli in the cases.

The treatment is similar to that for other forms of sycosis. Hyde and Montgomery have had good results from the use of x-rays.

ULERYTHEMA OPHRYOGENES (Taenzer) is a condition similar to, if not identical with, folliculitis decalvans, but in the eyebrows and face.

DEPILATING FOLLICULITIS OF THE LIMBS.—Under this name Arnozan has described a condition similar to folliculitis decalvans, which occurs on the hairy surfaces of the legs and thighs.

FOLLICULITIS NECROTICA is probably the same condition, on the trunk.

CUTIS VERTICIS GYRATA (Vereiss, *Derm. Zeitschr.*, B. xv, 11).—This condition, to which Jadassohn first called attention, consists in a peculiar conformation of the scalp which causes the skin of the vertex and back of the head to be thrown into furrows, producing an appearance suggesting the sulci over the surface of the brain. The condition gives rise to no symptoms and is apparently due to some preceding chronic inflammatory process of the scalp, resulting in atrophic lines between which furrows of unaltered skin project.

XVII.—SYCOSIS

SYNONYMS: *Sycosis vulgaris, Nonparasitic sycosis, Folliculitis barbæ, Acne mentagra, Sycosis staphylogenes, Barber's itch.*

Sycosis is a pustular folliculitis of the hair follicles of the beard and mustache. It occurs in two forms: sycosis vulgaris, due to infection with the common pus organisms, and tinea sycosis due to a combined infection with ringworm fungi and common pus organisms. (See Ringworm.)

Common sycosis was formerly described, in contradistinction to tinea sycosis, as nonparasitic sycosis, a title which is, of course, inappropriate.

Symptomatology.—The lesions of sycosis are inflammatory papules or pustules, which occur around hair follicles and are pierced by hairs. The lesions may remain discrete, but usually are confluent in irregular patches. They vary from rather superficial papules and pustules to deep, follicular abscesses. In the papular lesions the hairs may be firmly attached; usually, however, they are partly loosened, and when they are extracted the sheaths of the follicles are apt to come with them. In the pustular lesions the hairs are usually quite loose, and come out without forcible traction. The follicular abscesses may or may not destroy the follicles. In the cases of moderate severity there is not likely to result any perceptible thinning of the hairs, but in the severe, long-standing cases there may be produced partial destruction of the beard. According to the variations in its features, the condition shows various degrees of intensity and extent. The general picture is that of a pustular folliculitis, manifestly involving the hair follicles and sharply confined to the bearded parts of the face. Where the lesions are abundant and confluent, irregular, suppurating areas are produced, upon which pus accumulates and dries

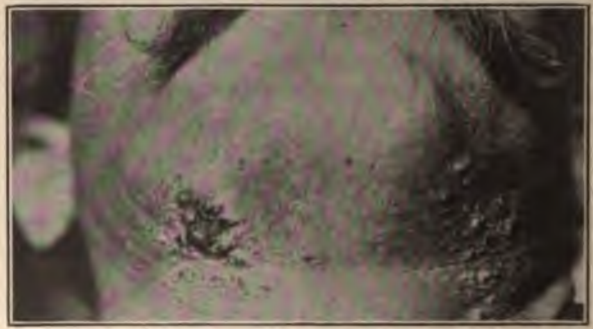

FIG. 341.—SIMPLE SYCOSIS. (Author's collection.)

into dirty, yellowish crusts. The disease may involve the entire mustache and beard. In such cases there is great temporary disfigurement, but the hypertrophic granulations which may be so marked in tinea sycosis are lacking.

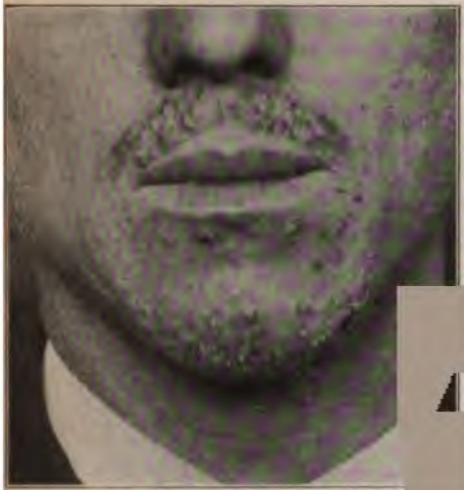

FIG. 342.—SIMPLE SYCOSIS. (Author's collection.)

As a rule, the disease is confined to patches, or to more or less discrete groups of lesions involving only part of the bearded area of the face. Common sycosis attacks the upper lip and the bearded part of the face indiscriminately. The upper lip is rarely uninvolved in prolonged cases, in this particular differing from tinea sycosis, which rarely involves the upper lip. The disease, however, may begin at any point where infection occurs. As a matter of fact, a pustular folliculitis in coarse hairs on the other parts of the body may occur of identically the same character as sycosis.

The disease pursues a persistent course. Follicles constantly become infected, and the disease is thus renewed by the appearance of new lesions, while, because of the deep seat of the organisms in the tissues, the old lesions are very slow to disappear. Recurrences are very likely to occur after treatment unless the results are radical.

Etiology and Pathology.—Sycosis is due to infection with the common pus organisms, with staphylococcus aureus and staphylococcus albus, which gain access to the tissues through the hair follicles and excite a perifolliculitis. It is conceivable, of course, that the disease might be produced by other pus-forming organisms, and Tommasoli has described a case in which it was due to a bacillus.[1]

Exciting causes are the various conditions which expose the parts to infection. The disease is most frequent in those who are careless in their habits or whose occupations expose them to dirt. A very frequent cause of the disease is the infection of the upper lip through nasal secretions. In some cases it starts in wounds produced by shaving.

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[1] Cronquist (*Archiv*, vol. lxxx, No. 1, 1906) reports a case of gonorrheal inflammation of the hair follicles of the skin of the abdomen in a woman twenty-one years old who was suffering at the same time from a vaginal gonorrhœa — Wright (*Jour. A. M. A.*, 1909, vol. lii, p. 1996), has had a sycosis due to gonococci contracted from a barber with gonorrhea.—Preis, *Archiv*, 1908, B. 92, p. 205 (agminate folliculitis from acid-fast bacilli).

Anatomically the lesions consist of a perifollicular inflammatory process which may or may not go on to abscess formation with the abscess cavities centering in the follicles. The inflammatory process may involve only the upper part of the follicles, or it may involve their entire depth and cause their complete destruction.

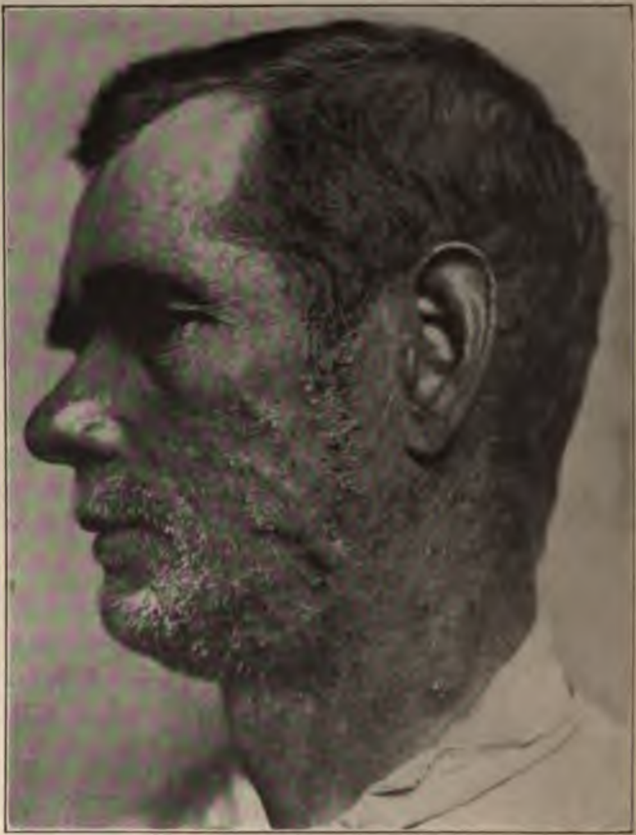

FIG. 343.—SIMPLE SYCOSIS. (Hartzell's collection.)

Diagnosis.—The disease is most likely to be confused with pustular eczema or impetigo involving the bearded part of the face. In these the inflammatory process is more superficial, is not invariably centered around the hair follicles, there is no loosening of the hairs, and the disease does not limit itself to the hairy surfaces.

Tinea sycosis produces a much more angry picture. There is the formation of hypertrophic frambœsiforme masses with abundant suppuration, the process extends deeply into the tissues, and greater scarring results. It rarely involves the upper lip. The diagnosis, of course, should be confirmed in tinea sycosis by examination for the fungus. In pustular syphilides of the face the eruption does not confine itself to the bearded part of the face, and there are always other evidences of syphilis. Some of the cases of lupoid sycosis are indistinguishable from simple sycosis developing on the cheeks, if, indeed, these cases of lupoid sycosis have any essential distinction from common sycosis.

Prognosis and Treatment.—The disease is extremely obstinate, and requires persistent treatment in order to cure it. Its treatment has been rendered much simpler and more radical by the use of Röntgen therapy.

The treatment may be said to be entirely local, although, of course, in patients who are below par everything that tends to increase the physical vigor increases the ability to resist the infectious process. The local treatment is directed to the removal of the infecting organisms. In the beginning of treatment the first measure is to remove pus crusts from the surface. This is readily done, after clipping the hairs short, by the application of boric-acid wet dressings or other nonirritating wet dressings, or by the use of boric-acid and vaseline ointment, or an ointment of precipitated sulphur, two or three per cent, or ammoniated mercury, two or three per cent, in vaseline. Before the application of any of these ointments the surface should be cleansed by bathing, with the use of soap if it is not too irritable, and the ointment then freely applied.

After the surface has been cleansed by the use of these measures for a day or two, and after the inflammation has subsided somewhat in the acute cases, one proceeds to the more vigorous antiseptic measures. In the first place, it is necessary to keep the hair short. It is best to shave the parts every other day. If this is too painful at first, the hair can be clipped short with a pair of scissors, but shaving is better. Loose hairs should be epilated from day to day, but this epilation should not include the firmly attached hairs; with such hairs it is very painful, and the benefit is relatively slight. It is my practice to proceed from this point in the treatment of these cases with the use of x-rays, keeping the hair clipped and the surface free from pus by the usual measures. Where x-rays are not to be used, the further treatment requires the application of vigorous antiseptics. Under such conditions the most satisfactory course to pursue is to evacuate each follicular abscess as it forms by pulling out the hair; then the point is touched with a strong, penetrating antiseptic, like a solution of equal parts carbolic acid, tincture of iodine, and chloral hydrate. This mixture may also be applied to papular lesions, but of course it cannot be used too freely at one time. In conjunction with this the patient uses an ammoniated mercury or sulphur ointment of from two- to ten-per-cent strength. The ointment is to be applied freely at night, and where irritation is not too great it should be rubbed in, with a view to causing some penetration into the follicles. These applications are combined with frequent shaving and the daily use of antiseptic washes, to keep the surfaces free from pus.

The number of other agents which may be used includes almost all antiseptics. One of the very good applications is Vleminckx's solution, used in the very same way as in acne. Other agents which are more or less frequently used are oleate of mercury, from four to twenty per cent in vaseline; resorcin, from five to ten per cent in ointment; ichthyol (a poor application because of its disagreeable odor and color), from five to twenty per cent in ointment.

The treatment of cases with Röntgen rays is like that of acne. Parts not diseased are, of course, protected by lead foil. The treatment may be carried out a little more vigorously than in acne, but it is not necessary to produce a marked erythema. It may be necessary, however, to carry the treatment to the point of causing out-fall of the hairs. When this occurs there is usually an end of the sycosis. From a considerable experience in the treatment of sycosis and tinea sycosis with Röntgen therapy I can recommend it strongly. The cases yield much more readily than from any other method, and the results are radical. After the cases are symptomatically cured with x-rays they show little tendency to recur.

XVIII.—DERMATITIS PAPILLARIS CAPILLITII[1]

SYNONYMS: *Acne keloid* (Bazin), *Sycosis nuchæ necrotisans* (Ehrmann), *Sycosis papillomateux and Sycosis frambæsiformis* (Hebra).

Under this name is described a pustular folliculitis which occurs at the hair line upon the back of the neck, and is accompanied by hypertrophic scarring.

Symptomatology.—Clinically the affection has the appearance of a deep-seated, pustular folliculitis in the hair follicles on the back of the neck. It is chronic, and there form hypertrophic granulations which are followed by keloidal scars, the process resulting in the formation of a roughened, nodular, or papillomatous area. The lesions vary from acutely inflamed follicular papules and pustules to nodules the size of a pea or perhaps larger. Some of the nodules are masses of hypertrophic scar tissue, which may be white and without active inflammatory changes; as a rule, the lesions are pink to red, often with a purplish tinge, and follicular abscesses in them are common. There may be a good deal of undermining of the lesions with pus, and the purulent secretion may be sufficient to give the diseased area a disgusting odor. The process destroys the hair follicles and produces a partial baldness, but a part of the follicles escape and irregularly placed hairs usually stud the diseased area. The disease begins as a pustular folliculitis, and the hypertrophic scarring is a gradual evolution. The course is chronic, with little tendency to get well because of the deep infection of the hair follicles. The diseased area usually remains confined to the nucha, but it may spread upward as far as the vertex.

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[1] Guszman, *Dermatolog. Zeitschrift*, 1905, No. 3.

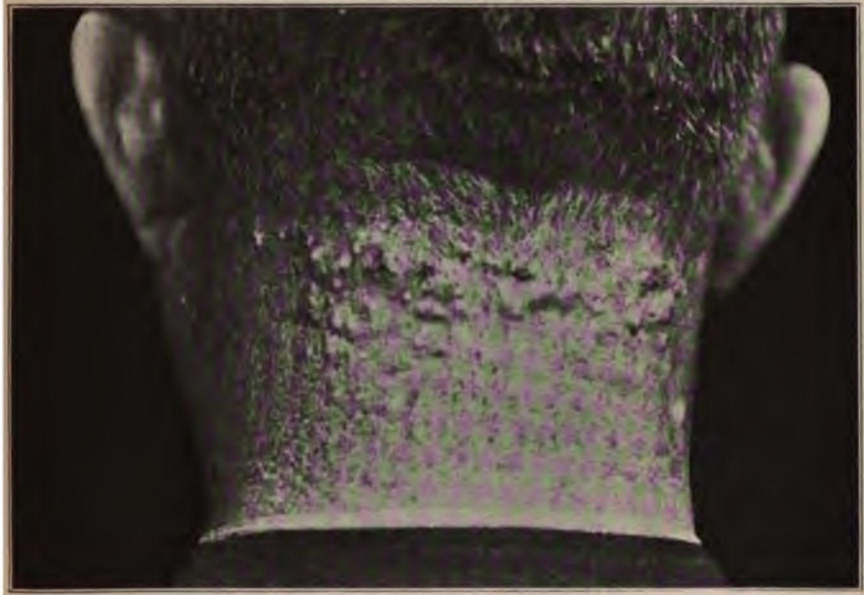

FIG. 344.—DERMATITIS PAPILLARIS CAPILLITII. (Author's collection.)

Etiology and Pathology.—Well-marked cases are said to be rare, but in my experience the condition is by no means a curiosity, particularly in negroes. It is most common in men, and develops during adult life.

There has been considerable discussion as to the character of the disease. From the cases which I have studied I have no doubt that it is a pustular folliculitis of the hair follicles due to the common pus organisms, and because of its chronicity followed by exuberant granulations and hypertrophic scars. A few hypertrophic scars on the back of the neck from follicular abscesses are by no means an uncommon condition. In the cases there is frequently a history of having begun in boils on the back of the neck.

Diagnosis.—The location and the hypertrophic scarring are so characteristic that there should be no difficulty in diagnosis. There is no closely similar condition.

Prognosis and Treatment.—As in deep-seated sycosis, the treatment is difficult, and apt to be unsuccessful unless persistently carried out.

The treatment is like that of sycosis. Diseased hairs should be epilated, the surface freed of pus, follicular abscesses opened, emptied, and swabbed out with carbolic acid or some other strong antiseptic, and the entire area should be kept clean by the use of an antiseptic application, such as weak bichloride wet dressings, or frequent washing with bichloride solution. The most satisfactory treatment is by the use of x-rays, as in sycosis. With that agent the disease can be eradicated and the hypertrophic scarring very much reduced.

D. DISEASE OF THE NAILS[1]

The only diseases of the nail in which the nail plates are primarily involved are those in which the nails are invaded by the fungi of ringworm and favus. The other alterations of the nails occur secondarily to disturbances of the underlying tissues. Indeed, excepting the conditions in which the nails and the adjacent tissues are involved directly by the attacks of microörganisms or by injury, all of the so-called diseases of the nails are but the nail symptoms of underlying conditions. The nails are especially sensitive to nutritional disturbances. This is perhaps largely due to the fact that they are situated at the most distant terminal areas of circulation, and are thus most readily affected by circulatory disturbances. In addition to this, however, they are also extremely sensitive to trophic disturbances which leave traces in the growth of the nails. These secondary changes in the nail from general disturbances of the health furnish an interesting field of observation, all the more so because they are capricious in their occurrence and often difficult of explanation.

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[1] Shoemaker, *Jour. Cutan. Dis.*, 1890.—Hutchinson, *Arch. of Surg.*, 1891, p. 237.—Heller, "Die Krankheiten der Nägel," Berlin, 1900 (complete monograph).—Hardaway, *Jour. Cutan. Dis.*, 1901, p. 522.—Grindon, *Jour. Cutan. Dis.*, 1901, p. 516.—Pollitzer, *Jour. Cutan. Dis.*, 1901, p. 503.—C. J. White, *Boston Med. and Surg. Jour.*, November 13, 1902.—Leviseur, *Jour. Cutan. Dis.*, 1902, p. 502.—Jackson, *Jour. Cutan. Dis.*, 1905, p. 151.—Nobl, *Wien. klin. Rundsch.*, 1905, No. 27.

HYPERTROPHY OF THE NAILS

I.—ONYCHAUXIS

ETYMOLOGY: ὄνυξ, nail, and αὔξειν, to grow.

Onychauxis is overgrowth of the nail plate. It may be simple enlargement of the nails without other changes, but in most cases it is the result of nutritional causes producing changes in the texture, color, and shape of the nails.

Symptomatology.—Simple enlargement of the nails is seen in various conditions like acromegaly, in which the distal phalanges become enlarged, the nails sharing the general increase in size. The most familiar illustration of simple hypertrophy of the nails is *hippocratic nails*—the large, rounded nails which occur on the bulbous or spade-shaped finger tips that develop in certain chronic diseases, particularly in chronic pulmonary and cardiac diseases. Hippocratic fingers usually develop slowly and involve both hands, but they may develop in a few weeks and be confined to one hand. Their occurrence is usually explained on the ground of nutritional disturbance arising from poor peripheral circulatior. In my opinion this explanation is not satisfactory in many cases. It seems more likely that the changes are essentially trophoneurotic rather than circulatory, but why they should occur especially in pulmonary and cardiac diseases we do not know.

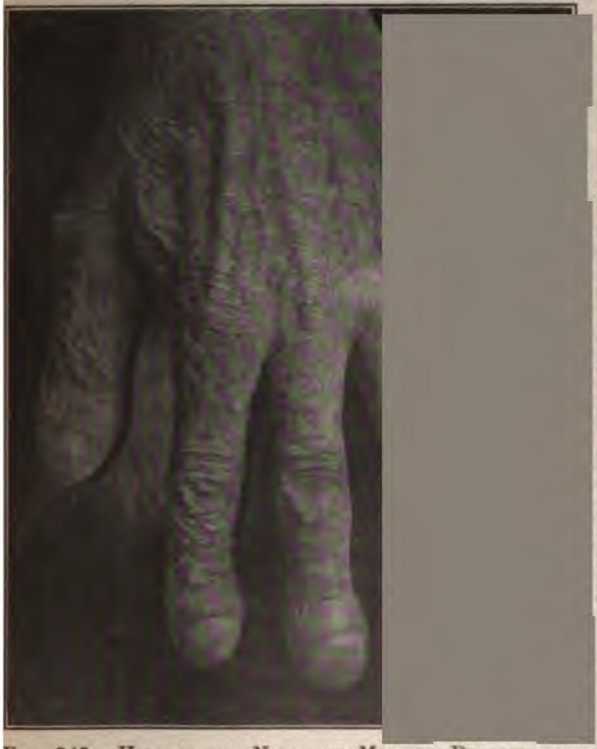

FIG. 345.—HIPPOCRATIC NAILS OF MARKED DEGREE, AND VERY UNUSUAL IN THAT IT INVOLVED ONLY ONE HAND. (Cook County Hospital case.)

In very rare cases simple enlargement of the nails occurs without other changes in the finger tips. Keyes has described a case in which the nails extended around the tips of the fingers and toes, and forward beyond the extremities. The texture of the nails was normal, and the condition was congenital. In the usual form of onychauxis the nails become thickened, softer than normal, more or less roughened, and lose their luster, and become yellowish to dirty brown in color. There is often imperfect cornification of the nail plate, and the accumulation beneath it of soft, horny epidermis. This is particularly likely to occur at the free border of the nail, where the nail may be lifted up and separated from the matrix. The nails are apt to be brittle, so that their edges are jagged and cracked. The condition may involve all or only a few nails. The toes are most frequently affected in onychauxis, particularly the big toes.

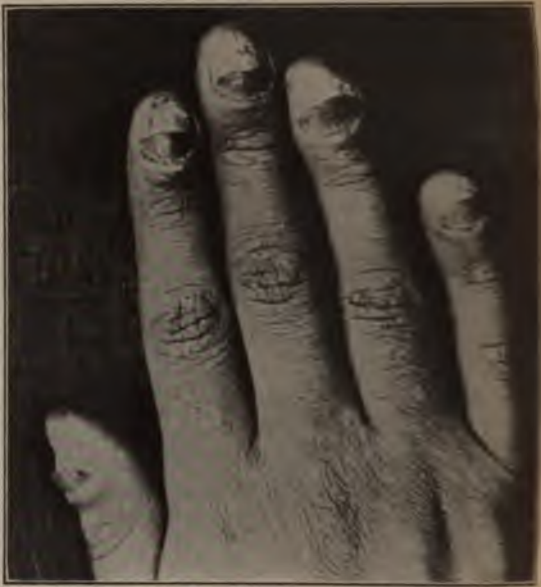
FIG. 346.—PSORIASIS OF NAILS.
(C. J. White's collection.)

Onychogryphosis (ὄνυξ and γρυπός, curved).—This is simply hypertrophy of the nail analogous to the common forms of onychauxis, but characterized by unusual increase in length, so that the nail projects beyond the digit, and is usually curved and twisted. In the extreme cases, where the nail is greatly thickened, rough, and blackish, and curved, it may have a fanciful resemblance to a ram's horn. It most frequently occurs on the great toe.

Ingrowing toe nail consists of the lateral growth into the soft parts of the border of the nail, either from a spontaneous downgrowth of the borders of the nail at the free edge or, more frequently, as result of pressure forcing the tissues against the border of the nail. It sets up paronychia, and is a surgical condition.

Etiology.—Onychauxis is in some cases a developmental defect, in some others it is the result of circulatory disturbances. In most cases it is the result of local irritative conditions; probably pressure and other mechanical causes account for most examples in the toes as well as many on the fingers. It may also result from many pathological processes involving the nails, especially eczema, psoriasis, ringworm, and favus. Occasionally it results from the rarer inflammatory dermatoses, like dermatitis exfoliativa, and pityriasis rubra pilaris, but it is rarely, if ever, produced by seborrhœic eczema.

The causes of onychogryphosis are the same as those of onychauxis. The great overgrowth in the length of the nail is usually due to the fact that the individual either will not or cannot keep the nails trimmed.

Diagnosis.—The forms of dermatoses producing onychauxis are not characteristic. An onychauxis, for instance, produced by ringworm cannot be distinguished by its appearance from one produced by eczema. The determination of the cause of onychauxis in such cases depends upon the discovery of other features of the exciting dermatosis.

Treatment.—The local treatment consists in mechanical measures to destroy the excess of nail. This may be done by liquor potassæ or sapo viridis, in the way described under onychomycosis, but for most cases salicylic acid is the best keratolytic agent. This may be used in the strength of from 20 to 60 grains to the ounce of vaseline and applied under a dressing to the nail until softening occurs; then the nail is pared down or smoothed by rubbing. Shoemaker highly recommends an ointment of oleate of tin, from 20 to 60 grains to the ounce of lard.

II.—ONYCHOMYCOSIS

ETYMOLOGY: ὄνυξ and μύκης, a fungus.

Onychomycosis is a disease of the nails produced by the invasion of ringworm or favus. The nails usually become thick, roughened, opaque,

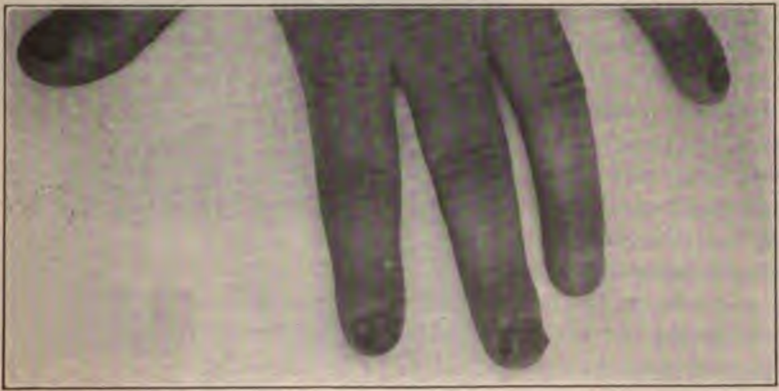

FIG. 347.—FAVUS OF NAILS. (C. J. White's collection.)

and brittle, as in onychauxis. One or more nails may be in part destroyed.

The diagnosis of onychomycosis depends upon the demonstration of the presence of the fungi. This is done by taking scrapings, or thin parings of the diseased nails and dissolving in liquor potassæ in the same way as in examining hairs for fungi; the discovery of the fungi is, however, more difficult than in the case of hairs.

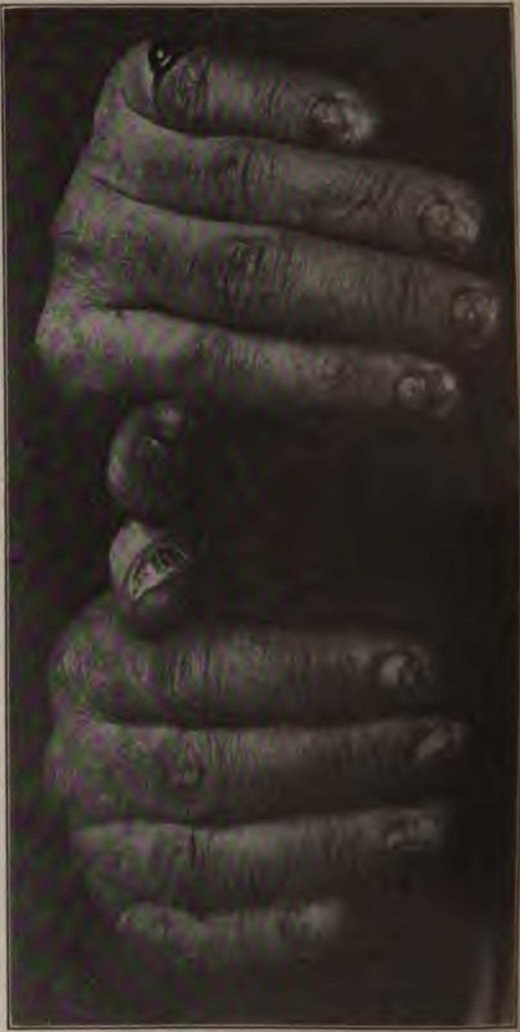

FIG. 348.—RINGWORM OF NAILS. (Engman and Mook's collection.)

In the treatment of onychomycosis the nails are first to be softened and dissolved away in part, and antiseptic applications then used. The readiest way of softening the nails is by the use of liquor potassæ, with which the nail is rubbed until as much of it is removed as is desired. Another way is by applying a dressing of green soap under rubber tissue, leaving this in position for a few days until the nail becomes soft. After the nail has been softened by either of these methods, strong antiseptics are used, such as corrosive sublimate from 1 to 2 grains to the ounce of alcohol, or wet dressings of sulphurous acid (fresh), or of hyposulphite of soda, twenty-five per cent. The treatment must be very persistent to be successful.

III.—SUPERNUMERARY NAILS

Congenital supernumerary nails are occasionally found, usually growing on the digits, very rarely in some abnormal position. In rare cases nails are also observed to develop on stumps of amputated digits.

IV.—PTERYGIUM

This, as applied to the nails, is the outgrowth of the posterior nail fold and its adherence to the nail plate. It is usually due to lack of care in keeping the nail fold loosened, and if neglected is followed by fissuring.

Retraction of the posterior nail fold, an opposite condition to pterygium, which results in exposure of the root of the nail (ficus unguium), is sometimes seen. It is probably the result in most cases of local causes.

ATROPHY OF THE NAILS

Atrophic changes in the nails as the result of circulatory or general nutritional disturbances are common. The nails may become thin, ridged, or furrowed, pitted or brittle, and these various changes may be combined in various ways.

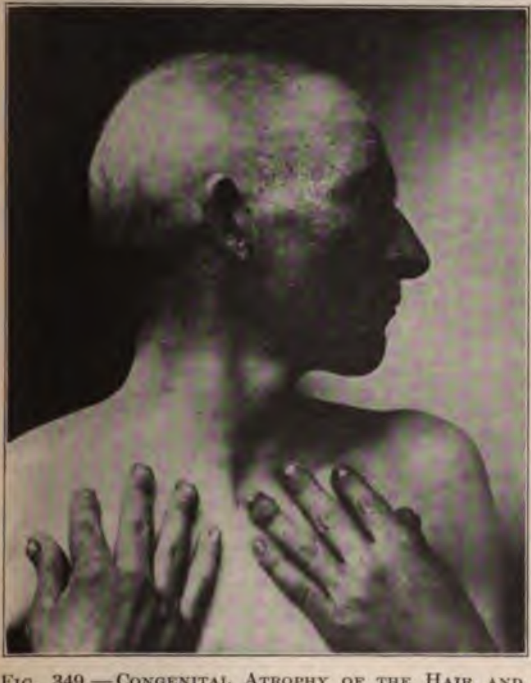

FIG. 349.—CONGENITAL ATROPHY OF THE HAIR AND NAILS. (C. J. White's collection.)

V. Congenital Atrophy of the Nails.—Congenital absence or atrophy of the nails occurs in rare instances. It is often associated with other developmental defects in the hair and teeth.

VI. Thinning of the Nails.—The nails may show atrophy simply by marked thinning of the nail plate without other changes. Most frequently it is associated with transverse furrowing or flattening of the nail plate, and at times some raising of the lateral borders so that the outer surface of the nails is in part curved, giving rise to the condition known as *spoon nails*. In many cases the thinning is accompanied by unusual brittleness (*onychorrhexis*), so that the free edge is fissured and broken. In most cases the condition is the result of nutritional disturbances, but at times no cause is ascertainable. In some cases the condition is hereditary.

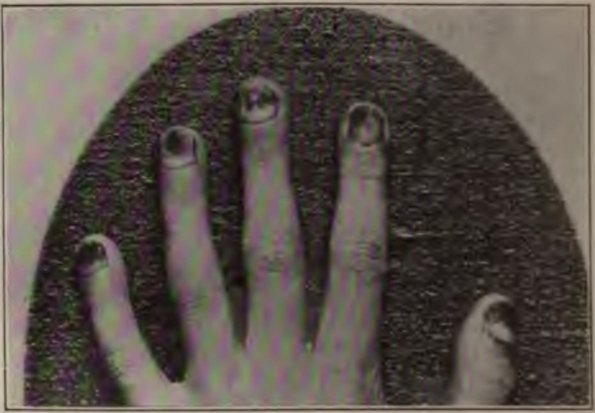

FIG. 350.—ATROPHY OF NAILS IN ALOPECIA AREATA.
(Grover W. Wende's collection.)

VII. Eggshell Nails.[1]—This condition, to which Hyde has called attention, is as follows: The nails are thin, with a distinct tendency to upward curving and separation of the nail plate from the matrix at the free border. Where separated from the nail bed the nail has the characteristic purplish-white hue of the inner surface of the shell of a hen's egg.

The condition is due to peripheral vascular disturbances, and Hyde has seen it only in women who were not in vigorous health.

VIII. Transverse furrowing of the nails is a very common manifestation of severe illness, the furrows representing temporary nutritional disturbances in the nails during the illness. Such furrows are very frequently seen as result of typhoid fever, and if there are relapses new furrows may be produced, so that until the nail grows out the attacks are recorded in these furrows.

IX. Ridged Nails.—Longitudinal, parallel ridges sometimes appear on the nails as exaggerations of the normal longitudinal striations on the nails. They are produced by shrinking of the nail substance between the normal striæ. They result from the same changes which produce other atrophic changes in the nails.

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[1] Hyde, *Jour. Cutan. Dis.*, April, 1906.

X. Separation of the Nails.—Separation of the nails from the matrix at the free border is sometimes seen. It may be the result of traumatism or of inflammatory processes. A slight degree of it is not uncommon in children, where no cause may be ascertainable. In some cases it is associated with neurotic disturbances.

XI. Shedding of the Nails.—Shedding of the nails is not uncommon as result of inflammatory processes, especially from involvement of the nails in severe dermatoses, like pemphigus foliaceus, pityriasis rubra, scarlatiniform erythema, and, occasionally, scarlatina. The nails may also be shed as result of trophic disturbances. It may involve one or all of the nails, most frequently that of the big toe. This sometimes occurs in syphilis, in acute infectious diseases, in diabetes mellitus, in alopecia areata, and in locomotor ataxia. It has been observed after section of the sciatic nerve, and in severe hysteria. It is seen in cases of annual keratolysis. Cases have also been recorded of annual shedding of the nails without keratolysis (Dubreuilh and Hilbert). D. W. Montgomery [1] has recorded a case in which there was continuous shedding of the nails, the process involving one nail after another and continuing indefinitely. The condition was hereditary.

XII. Leuconychia—(*Whitening of the Nails.*)—This may occur either in spots (leuconychia punctata), in transverse, parallel lines (leuconychia striata), or be total (leuconychia totalis). In most cases white spots in the nails are due to the presence of air between the layers of the nail, but in some cases there may be a pigmentary disturbance, for Unna has recorded a case of total leuconychia in which there was also a condition of ringed hairs. In most cases white discolorations of the nails are due to traumatism. In Heidingsfeld's case of leuconychia striata the white bands were discovered to be due to cutting and pressing back the nail fold. In some cases, however, punctate leuconychia seems to be undoubtedly a trophic disturbance.

XIII.—ONYCHIA (Onychitis)

Symptomatology.—Onychia is inflammation of the nail matrix. It is usually acute, but may be chronic. The condition shows the changes of inflammation in the vascular structures beneath the nail. In the acute cases there is redness or lividity, tension, and throbbing pain. Suppuration usually follows; the nail is separated from the matrix, becomes thickened and opaque, and is usually thrown off, leaving a granulating surface upon which a stunted nail re-forms. In the subacute cases, as from syphilis, hypertrophic granulations form under the nail, usually beneath the free border. The nails are partially separated, become opaque, roughened, and may or may not be completely shed. If thrown off, the new nail is usually deformed.

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[1] *Jour. Cutan. Dis.*, 1897, p. 374.

Etiology.—Onychia is most frequently due to infection or traumatism. It is particularly likely to occur in strumous children in the form of acute onychia resulting from pressure or from bruises. This is the so-called *onychia maligna.*

Onychia may also result from the involvement of the nail matrix by syphilitic eruptions. In one form of *syphilitic onychia,* the onychia sicca, the changes are those of atrophy of the nail without inflammation. This is not really a syphilitic onychia, but simply an atrophy of the nails such as occurs in other diseases. The nails become roughened, fissured, or pitted, lose their luster, and become brittle. In other cases syphilitic papules or tubercles form under the nail, lift up its free border, and cause more or less destruction of the nail plate. The nails may become hypertrophied or roughened, or one or more nails may be in part or entirely destroyed. In such cases the new nails are likely to be deformed or rudimentary.

Treatment.—Onychias of specific type require rational constitutional treatment. The local treatment of onychia is surgical. Provision must be made by incisions for escape of pus and the lesions dressed with antiseptic applications, preferably wet dressings. In the early stage of acute onychias, cold wet dressings give great relief from pain.

SECTION XV

DISEASES OF MUCOUS MEMBRANES[1]

As has been seen, the mucous membranes, especially of the mouth, are frequently involved in many dermatoses. These are for the most part dermatoses of constitutional origin, like erythema multiforme, dermatitis herpetiformis, pemphigus, pemphigus foliaceus, syphilis, lichen planus, and general exfoliative dermatitis.

It is not the purpose of this chapter to take up the mucous membrane lesions of these various diseases, but to consider a few conditions confined to the mouth which usually come under the observation of the dermatologist.

I.—LEUKOPLAKIA

SYNONYMS: ***Leukoplakia buccalis, Leukokeratosis buccalis, Leukoplasia, Ichthyosis linguæ, Tylosis linguæ, Psoriasis of the Tongue, Buccalis.***

Leukoplakia buccalis is a disease of the mucous membrane of the mouth characterized by the formation of one or more whitish plaques which are persistent and tend to increase in thickness.

Symptomatology.—Leukoplakia in its slightest manifestation occurs as a minute, whitish plaque of slightly thickened horny epidermis. The disease may consist of only one plaque, but in many cases the plaques are multiple and often coalesce into patches. These patches are of irregular shape, and usually of ill-defined outline and very slightly thickened. The condition may remain of this superficial type for many years, or it may disappear spontaneously. If it persists the hyperkeratosis tends to increase, and after many years, or sooner in extreme cases, the patches, particularly at their centers, where the hyperkeratosis is greatest, may become dead white and studded with minute, papillated elevations. As a rule, the affection is limited to a few discrete or irregular confluent patches, but in some cases the condition is very extensive and may involve almost the entire oral mucous membrane.

The lesions occur most frequently on the buccal mucous membrane just within the commissures of the mouth, or along the line of contact of the upper and lower teeth, but they may occur anywhere upon the buccal or lingual mucous membrane. As a matter of fact, they may occur on other mucous membranes. Lesions of the same character are sometimes seen on the vulva and penis, and may be followed by epithelioma. (Cf. kraurosis vulvæ.) The condition is without subjective sensations. Its importance lies in the fact that it is very frequently the site of cancerous degeneration. When that occurs the lesions become first irritable and inflamed, and then frank infiltrations of epithelioma appear.

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[1] **Fordyce, *Jour. Cutan. Dis.*, September, 1904.—Engman, *Jour. Cutan. Dis.*, 1904, p. 412.**

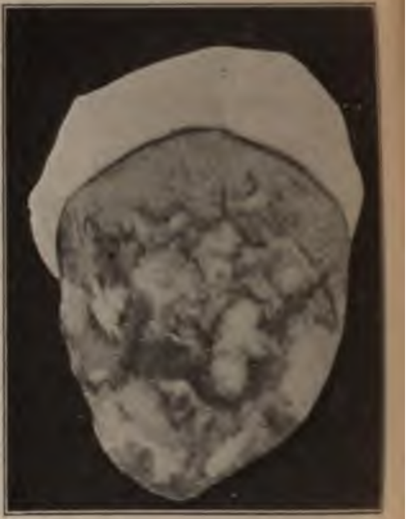

FIG. 351.—LEUKOPLAKIA LINGUÆ. (Reproduction in black and white from Jacobi's dermachromes. By permission of The Rebman Company.)

Etiology and Pathology.—The etiology of the condition is not known beyond the facts that long-continued irritation and syphilis are predisposing causes. Excessive smoking is perhaps the most important source of irritation in producing the lesions. Irritation from rough teeth may also excite it. The disease is seen often in persons who have had syphilis, but it is very frequently seen also in those who have not had it. Numerous cases have been observed in patients who have subsequently contracted syphilis, and the disease is undoubtedly not always parasyphilitic. There is, indeed, reason to question whether syphilis is a factor in its causation.

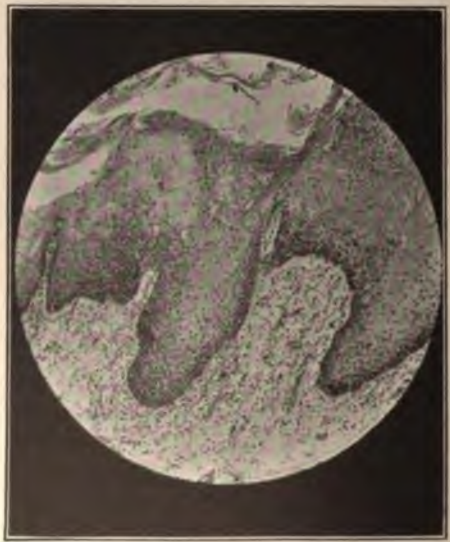

FIG. 352.—LEUKOPLAKIA OF TONGUE. (Fordyce's collection.)

Anatomically the changes at first are those of hyperkeratosis with downgrowth of the rete pegs and inflammatory changes in the subepithelial tissues. There may be in places obliteration of the papillæ, but more frequently there is exaggeration of the interpapillary epithelial processes, producing a microscopical picture closely simulating that of tubular epithelioma. When the growths become malignant they show the features of tubular epithelioma.

Diagnosis.—The characteristic feature is the presence of one or more persistent, whitish patches showing more or less hyperkeratosis. It can only be confused with subacute inflammatory lesions on the mucous membranes in connection with inflammatory dermatoses, like lichen planus. In such cases the association with cutaneous lesions gives the clue to the diagnosis. If cutaneous lesions are lacking a differential diagnosis from leukoplakia might be impossible.

Prognosis and Treatment.—Occasionally the patches will disappear spontaneously upon stopping smoking or correcting other sources of irritation. As a rule, however, the patches are permanent and are very intractable to treatment.

In the management of the cases all sources of irritation should be removed. The patients had better stop smoking entirely. The teeth should be looked after, the use of hot drinks, of highly seasoned, and therefore irritating, foods and of alcohol, should be avoided. In addition to these general measures the condition may be treated with soothing applications, or the patches may be radically destroyed. Occasionally the patches are much improved or disappear under the daily use of a bland mouth wash, like Dobell's solution, or a saturated solution of boric acid with a few drops of oil of cassia to the pint, combined with daily application to the patches of tincture of myrrh, or tincture of benzoin, or Peruvian balsam. The thicker patches can only be removed by destruction. I have succeeded in curing a good many of them by the use of x-rays. Next to this the best method of treatment is the destruction of the patches with the actual cautery. Other methods of destroying them are by the application of liquor hydrargyri nitratis, or twenty-per-cent chromic-acid solution. In the application of these solutions the parts must be thoroughly dried, surrounding parts protected by cotton, and the action of the caustic limited to the patch. Cauterization should be vigorous enough to cause destruction of the patch. The use of superficial caustics, like silver nitrate or carbolic acid, is harmful.

II.—TRANSITORY BENIGN PLAQUES OF THE TONGUE[1]

SYNONYMS: *Exfoliatio areata linguæ, Pityriasis linguæ, Annulus migrans, Glossitis areata exfoliativa, Erythema migrans, Wandering rash, Ringwormlike Patches of the Tongue, Circinate Eruption of the Tongue.*

Symptomatology.—The condition consists of spreading, scaly patches which appear upon the dorsal surface of the tongue. A patch begins as a slightly scaling macule on a red base, and this lesion spreads peripherally, with a tendency to involution in the center. As the lesion gets larger the center approximates the normal color of the epidermis but shows slight scaling, while the border may be red, or it may be whitish or yellowish from the abundant scaliness. The condition may consist of one or more plaques. These may reach the size of half an inch or even an inch in diameter, and when situated at the border of the tongue, where they are likely to occur, they usually spread only upon the dorsal surface forming a half circle. When the lesions are numerous they coalesce into gyrate figures (geographical tongue).

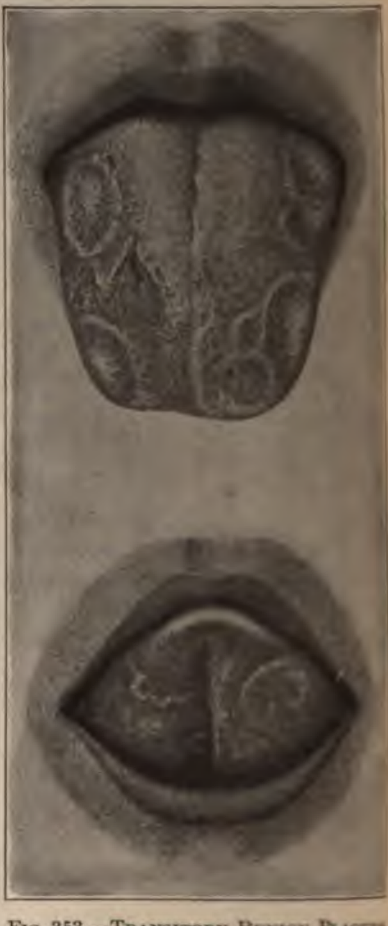
FIG. 353.—TRANSITORY BENIGN PLAQUES OF THE TONGUE. (Caspary.)

Occasionally a new central lesion may develop while the spreading border still exists, and thus two or three concentric patches may develop (iris form). Often the patches are limited to one area, or they may develop irregularly upon different parts of the tongue. Usually they are confined to the upper surface, but they may appear upon the lower surface of the tongue. The plaques are evanescent. They spread rapidly, and after one or two weeks the border fades out, leaving the mucous membrane of normal appearance. The condition, however, is continued by the reappearance of new patches. It is likely to be chronic. Patches may disappear, leaving the tongue free, and then recurrences develop irregularly, or the condition may continue indefinitely by the constant appearance of patches. The patches are without subjective symptoms as a rule, but in some cases there is itching.

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[1] Butlin, "Diseases of the Mouth," Cassell & Co., London, 1900.—Colcott Fox, *Lancet*, 1884, i, p. 842 (bibliography).—Kinnier, *Jour. Cutan. Dis.*, 1887, p. 56.—Hartzell, *Med. News*, vol. li, 1887.—Böhm, *Sammlung klin. Vorträge*, Leipzig, 1899, No. 249, p. 1467 (complete review).

The condition is rare, and is usually seen in children, most frequently under two years of age; it may appear, however, in adults. In a case under my observation the patient was a vigorous man of forty-five. Its cause is not known. It has been regarded as syphilitic (Parrot), and as parasyphilitic (Fournier), but in the opinion of most observers there is no reason to connect it with syphilis. Patients who had it have contracted syphilis. It is also attributed to gastrointestinal troubles, and this is a plausible explanation of its occurrence. Unna regards it as a trophoneurosis, and Besnier as related to seborrhœic eczema. The behavior of the affection suggests that it is parasitic, but except one case, in which a trichophytonlike fungus was found, no especial organism has been discovered. In some cases there is an hereditary tendency. It has been observed in delicate children, and to develop after acute illness. On the other hand, in many cases it occurs in vigorous individuals and without connection with any illness.

According to Parrot the condition is a superficial inflammatory process associated with parakeratosis. Unna, on the other hand, regards it as primarily a disease of the epithelium, a view which does not accord as well as Parrot's with the clinical manifestations.

Prognosis and Treatment.—The duration of the condition is altogether uncertain and is little influenced by treatment. The use of bland antiseptic mouth washes, and painting with such applications as tincture of myrrh or balsam of Peru, would seem to be indicated. Unna recommends sulphur washes. Hartzell and others believe that arsenic internally benefits the condition, while Stelwagon recommends attention to the gastrointestinal tract.

III.—BLACK TONGUE[1]

Synonyms: *Hairy Tongue, Lingua nigra, Hyperkeratosis linguæ.*

This condition consists in discolored patches on the tongue due to hyperkeratosis of the papillæ. The color is usually black, but it may vary from yellow to blue. Occasionally the papillæ are not apparently elongated, and the condition consists simply in slight thickening and blackening of their tips. More frequently, however, the papillæ are enormously lengthened so that they resemble hairs. The condition usually appears in the median line of the tongue in front of the circumvallate papillæ, and consists of one large patch, but it may be unsymmetrically situated and appear upon any part of the dorsal surface of the tongue.

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[1] Sendziak, *Monats. für Ohrenheilk*, 1894, vol. xxviii, p. 112 (review).—Goodale *Jour. Boston. Soc. Med. Sci.*, 1897–98, vol. ii p. 204 (histology).—Gottheil, *Arch Pediat.*, 1899, p. 255.

The duration of the condition is uncertain. It may disappear spontaneously in a few weeks, or it may persist for months or years. As a rule, it disappears spontaneously in time. Where the lengthening of the papillæ is marked there may be some disturbance of taste. Besides this the condition has no subjective symptoms, although in a few instances it has been painful. The disease is rare, and its cause is unknown. Its general characteristics suggest a parasitic cause, and spore-like bodies have been described by Gottheil and others, but no definite relation between the condition and any organism has been established. Anatomically the lesions consist of enormous lengthening of the filiform papillæ from hyperplasia and hyperkeratosis of the epithelium.

Treatment consists in cleanliness and the use of antiseptic mouth washes. Nothing is gained by scraping off or otherwise destroying the projecting papillæ.

IV.—FURROWED TONGUE

SYNONYMS: *Grooved Tongue, Wrinkled Tongue, Sulcated Tongue, Cleft Tongue, Fluted Tongue, Ribbed Tongue, Lingua plicata, Scrotal Tongue.*

This is a condition of unusual furrowing of the dorsal surface of the tongue. The central furrow is deepened, and radiating from this there are numerous parallel, deep furrows which may resemble roughly the branches of a tree. The tongue looks as though too large for its mucous covering. The appearance is most aptly compared to that of the skin of the scrotum when contracted by cold. There are no changes in the mucous membrane characteristic of it. Occasionally the sulci may be so deep that food lodges in them and causes irritation. The condition is often hereditary and in nearly all cases is congenital, although it does not usually develop until adolescence. It may also be produced by chronic glossi-

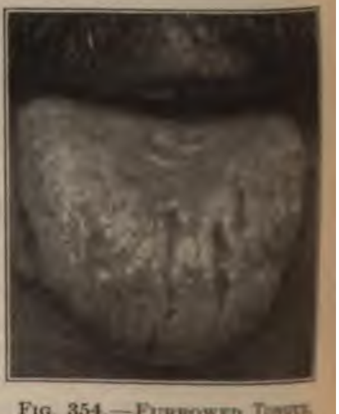

FIG. 354.—FURROWED TONGUE. (Author's collection.)

tis, especially when this results in macroglossia. It might be confused with scarring of the tongue from ulcerating gummata or tuberculous ulceration. In these the furrowing is irregular and limited in extent, the tongue is distinctly scarred, and there is a history of previous ulceration. The condition is permanent, but is the source of no trouble. Care, however, should be taken to prevent accumulation of food in the sulci and irritation from that cause.

V.—CHEILITIS GLANDULARIS

SYNONYMS: *Cheilitis glandularis apostematosa, Myxadenitis labialis.*

This is a condition characterized by dilatation of the mucous glands of the lips, accompanied by a free discharge of thick mucus and more or less inflammation of the mucous membrane. The glands become swollen to the size of a pinhead or larger, with dilated openings from which there is a mucous or muco-purulent secretion which is apt to glue together the lips during the night. The openings of the glands may be large enough to permit the entrance of a probe, and the dilatation of the glands may be great enough to produce shallow, fistulous tracts. The formation of abscesses in them may occur. There may be an accompanying inflammation involving the surface of the lips which may spread to the skin.

The disease is rare, and its cause is unknown. It was first described by Volkmann. The cases are rebellious to treatment. This consists chiefly in the use of local astringent and antiseptic applications. The cauterization of the follicles with nitrate of silver or carbolic acid would seem to be indicated.

VI.—FORDYCE'S DISEASE[1]

SYNONYM: *Pseudo-colloid of the Lips* (Crocker).

This condition, to which attention was first called by Fordyce, consists in the presence of minute whitish or yellowish bodies in the mucous membrane of the mouth and vermilion border of the lips. The bodies are pinpoint to pinhead size, of whitish or yellowish color, usually without elevation, and are made more prominent by stretching the surface. They may be without elevation or project slightly. They are found especially on the buccal mucous membrane along the line of the teeth, on the mucous and vermilion border of the lips. They are abundant and are symmetrically distributed on the two sides. Usually they are discrete, but they may be so abundant as to coalesce into plaques. They are without subjective symptoms.

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[1] Fordyce, *Jour. Cutan. Dis.*, 1896, p. 413.—D. W. Montgomery and Hay, "Sebaceous Glands in the Mucous Membrane of the Mouth," *Proceedings of the Tenth Annual Session of the Assoc. Amer. Anatomists*, 1897, p. 76.—C. J. White, *Jour. Cutan. Dis.*, March, 1905, p. 97 (complete review of subject with bibliography).

The condition is not rare. It is found most frequently in male adults, but it may exist in infancy. Fordyce originally believed that the lesions were due to granular degeneration of the cells of the mucous membrane, and C. J. White upholds this view.[1] D. W. Montgomery, Hay and others, however, have shown that the bodies are due to the marked development of sebaceous glands which are present in these surfaces. Whether their unusual development in these cases is due to embryonic peculiarities, or is acquired, is not settled.

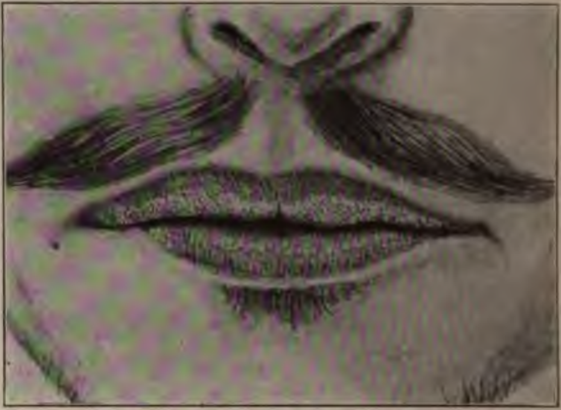

FIG. 355.—FORDYCE'S DISEASE. (Photograph of Fordyce's colored plate.)

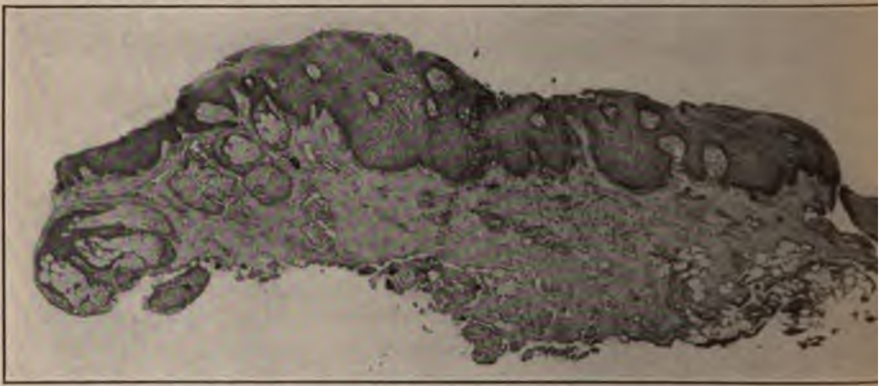

FIG. 356.—FORDYCE'S DISEASE. (C. J. White's collection.)

The condition is permanent, and requires no treatment.

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[1] From a study of 65 cases he concludes that: "The lesions of Fordyce's Disease owed their origin to the difficulties of digestion *with their subsequent toxæmias,* from which 50 out of the 65 patients suffered.")

VII.—PERLÈCHE[1]

Synonyms: *Bridou, Parasitic Disease of the Lips.* **Etymology:** *Perlecher,* to lick.

This is a condition characterized by the development at the commissures of the mouth of symmetrical, diphtherialike patches. It begins, as a rule, on the epithelium of the inside of the lips, and shows first as a whitening and scaling of the epithelium with some underlying hyperæmia. The epidermis becomes macerated and broken, the base is reddened, and there is weeping from the surface. The patches may thus become indistinguishable in appearance from syphilitic mucous patches. The patches on either side spread toward the median line, but do not involve the entire surface of the lip. They spread also upon the inner surface of the lip and to the adjacent skin. Fissures are apt to form at the commissures, thus increasing the resemblance to infantile mucous patches. There is a feeling of discomfort and dryness indicated by the constant moistening of the parts with the tongue.

The disease has been seen chiefly in France. It occurs in infants and young children, especially in institutions, and is contagious. It is thought to be transferred by immediate contagion through eating utensils and similar objects. Lemaistre, who first described it, believed it to be due to a specific organism called by him streptococcus plicatilis. Other observers believe that it may be produced by various organisms. Raymond believes it due in the majority of cases to the staphlococcus cereus albus. Sabourand says it is impetigo.

The disease is likely to be confused with mucous patches in infantile syphilis. In some cases the only way of making a differential diagnosis from mucous patches is by demonstrating the absence of other evidences of syphilis. It disappears in from a few weeks to one or two months, but shows a tendency to recur. Treatment consists in the use of mild antiseptic applications. Attention should be given to nursing bottles and other possible means of infection.

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[1] Raymond, *Bull. de la Soc. de dermat. et de syph.*, 1895, p. 289.—Planche *Thèse de Paris*, 1897.—Jacquet, *La Pratique*, T. 3, p. 839.

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